WHO/CDS/2005.26

COMMUNICABLE DISEASE TOOLKIT

SUDAN

World Health Organization

Communicable Disease Working Group on Emergencies, WHO/HQ
WHO Regional Office for the Eastern Mediterranean (EMRO)
WHO Country Office, Khartoum

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ANNEXES

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Further information is available at: CDS Information Resource Centre, World Health Organization, 1211 Geneva 27, Switzerland; fax: (+41) 22 791 4285, e-mail: cdsdocs@who.int

PREFACE

The purpose of this *Communicable Disease Toolkit for Sudan* is to provide ealth professionals in United Nations agencies, nongovernmental organizations, donor agencies and local authorities working in Sudan with up-to-date guidelines and standards for controlling communicable diseases.

The *Communicable Disease Profile for Sudan* aims to provide up-to-date information on the major communicable disease threats faced by the population. The list of endemic and epidemic-prone diseases has been selected on the basis of the burden of morbidity and mortality, and includes acute lower respiratory tract infections, African trypanosomiasis, bacillary dysentery, cholera, HIV/AIDS, malaria, measles, tuberculosis and yellow fever. Diseases for which there are global eradication or elimination goals are also included. The document outlines the burden of communicable diseases in Sudan for which data are available, provides data on recent outbreaks in the country and presents disease-specific guidelines on the prevention and control of these diseases.

The *Health surveillance forms* and *Case definitions* have been developed to provide early warning of epidemics, but will also monitor acute lower respiratory tract infections, sexually transmitted infections, injuries/trauma and malnutrition.

The *Guidelines for outbreak control*, *Case management of epidemic-prone diseases*, *Guidelines for collection of specimens for laboratory testing* and *Outbreak investigation kit* are aimed at facilitating outbreak preparedness and response.

The control of communicable diseases represents a major challenge to those providing health care services in Sudan and neighbouring countries. It is hoped that the *Communicable Disease Toolkit for Sudan* will facilitate the coordination of communicable disease control activities among all agencies working in this region.

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Edited by Dr Michelle Gayer, Dr Pamela Mbabazi, Dr Máire Connolly and Dr Albis Gabrielli of the Programme on Communicable Diseases in Complex Emergencies, WHO/CDS.

This Toolkit is a collaboration between the Communicable Disease Working Group on Emergencies (CD-WGE) at WHO/HQ, the Division of Communicable Disease Prevention and Control (DCD) at WHO/EMRO and the Office of the WHO Representative for Sudan. The CD-WGE provides technical and operational support on communicable disease issues to WHO Regional and Country Offices, ministries of health, other United Nations agencies, and nongovernmental and international organizations. This Working Group includes the Departments of Control, Prevention and Eradication (CPE), Surveillance and Response (CSR) in Communicable diseases (CDS), Roll Back Malaria (RBM), Stop TB (STB) and HIV/AIDS (HIV) in HTM; and the Departments of Child and Adolescent Health and Development (CAH), Immunization, Vaccines and Biologicals (IVB) and Health and Action in Crisis (HAC).

The following individuals at WHO/HQ, EMRO and the WHO Country Office in Khartoum contributed to the development of this document, and their technical input is gratefully acknowledged:

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COMMUNICABLE DISEASE TOOLKIT

SUDAN

1. COMMUNICABLE DISEASE PROFILE

World Health Organization
2005

Communicable Disease Working Group on Emergencies, WHO/HQ
WHO Regional Office for the Eastern Mediterranean, EMR0
WHO Country Office, Khartoum

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INTRODUCTION

The purpose of this document is to provide public health professionals working in Sudan with up-to-date information on the major communicable disease threats faced by the population. The list of endemic and epidemic-prone diseases has been selected on the basis of the burden of morbidity and mortality. Diseases for which there are global eradication or elimination goals are also included. The document outlines the burden of communicable diseases in Sudan for which data are available, provides data on recent outbreaks in the country and presents disease-specific guidelines on the prevention and control of these diseases.

The control of communicable diseases represents a major challenge to those providing health care services in Sudan. It is hoped that this document will facilitate the coordination of communicable disease control activities among all agencies working in the country.

1. ACUTE LOWER RESPIRATORY INFECTIONS (ALRI) – CHILDREN AGED UNDER 5 YEARS

DESCRIPTION

Infectious agent	Bacteria: the most common are likely to be *Streptococcus pneumoniae* and *Haemophilus influenzae* (and, to a lesser extent, *Staphylococcus aureus*). Several respiratory viruses.
Case definition	**Clinical case definition** "Pneumonia" is used at government health facilities as an action-oriented classification for management purposes according to both the ALRI and IMCI guidelines. It is therefore likely to include lower ARI clinically presenting with similar signs and symptoms, such as pneumonia, bronchiolitis and bronchopneumonia. The classification of cases aged under 5 years according to the national IMCI guidelines, which differ slightly from the ALRI guidelines, is as indicated below. **Children aged 2 months up to 5 years:** • **Pneumonia** *Symptoms:* Cough or difficult breathing; **and** *Signs:* 50 or more breaths per minute for infants aged 2 months up to 1 year, *or* 40 or more breaths per minute for children aged 1 up to 5 years old; **and** No general danger signs, chest indrawing or stridor in a calm child. • **Severe pneumonia or very severe disease** *Symptoms:* Cough or difficult breathing **and** any general danger signs or chest indrawing or stridor in a calm child. **General danger signs***:* unable to drink **or** breastfeed; vomits everything; convulsions; lethargic or unconscious. **Infants aged under 2 months:** **Severe cases** in young infants are classified broadly as "**Possible serious bacterial infection**", based on the presence of any of 16 signs or symptoms, among which are also respiratory signs such as fast breathing (60 or more breaths per minute), severe chest indrawing, nasal flaring, grunting and wheezing. Other signs include also fever or low body temperature, typical signs of infection (ear and skin), danger signs and feeding problems. **General danger signs***:* unable to drink or breastfeed; vomits everything; convulsions; lethargic or unconscious. Source: *National guidelines on Integrated Management of Childhood Illness – IMCI* (revised in 2001).
Mode of transmission	Airborne by droplet spread.
Incubation	Depends on the infective agent; usually 2–5 days.
Period of communicability	Depends on the infective agent; usually during the symptomatic phase.

EPIDEMIOLOGY

Burden	Pneumonia is reported as one of the leading causes of death in children aged under 5 years throughout the country. ➢ ALRI represented 20% of outpatient visits in the under-fives and were responsible for 41% of hospital admissions for the same age group in 1997, according to data from the Federal Ministry of Health. Pneumonia caused 16% of deaths in paediatric hospitals in 1996; acute respiratory infections were responsible for 19% of hospital deaths in the under-fives in 1997. Source: *Report on IMCI early implementation phase in Sudan.* Khartoum, Primary Health Care, Federal Ministry of Health, November 1999.
Geographical distribution	Throughout the country.
Seasonality	An ARI peak is likely to occur in the colder months (December–February).
Alert threshold	An increase in the number of cases above the level expected for the time of the year
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Influx of non-immune population into areas of new pathogen strains. War in Sudan has caused the displacement of significant numbers of people from war-torn areas to safer areas, including Khartoum. Crowded living conditions of internally displaced populations (IDPs) in the new areas may put them at higher risk of developing ARI.
Overcrowding	**Yes**	Overcrowding increases the risk of developing ALRI.
Poor access to health services	**Yes**	Access to services and drugs varies considerably between areas, especially in rural areas. High attrition rates of government health care providers, including those trained in child health (IMCI), are high and represent a major concern. Immunization coverage is low, with rates of 51% for measles, less than 50% for DPT3 and 27% for a fully immunized child in 2001 (Source for immunization rates: *Multiple indicator cluster survey*, Sudan, 2000). Prompt identification and treatment of cases by appropriate providers is the most important control measure. **Without proper treatment, the case-fatality rate is high (20% or more in emergency situations).**
Food shortages	**Yes**	Food insecurity is likely to occur in war-torn areas and among IDPs. Additional risk factors include: poor breastfeeding practices (less than 20% of infants aged under 4 months are exclusively breastfed), likely high malnutrition indicators (low birth weight, malnutrition, vitamin A deficiency) and poor feeding practices during illness (Source:*Multiple indicator cluster survey*, Sudan, 2000).
Lack of safe water and poor sanitation	**Yes**	Increased risk of ARLI is linked to inadequate hygiene and inadequate handwashing: 34% of the total population is using proper sanitary means of excreta disposal, the percentage being less than 25% for the poor households. Access to sources of safe water varies considerably, especially by standards of living, with the poor having very limited access to them. (Source : WHO/UNICEF *Multiple indicator cluster and Demographic and Health Surveys*. Sudan 2000: http://www.unicef.org/infobycountry/sudan_statistics.html).

Others	**Yes**	Indoor air pollution. Low temperatures may increase the relative risk of children's acquiring pneumonia.
Risk assessment conclusions		ALRI represent one of the major leading causes of death in children aged under 5 years in Sudan. Inadequate feeding practices, food insecurity and overcrowding among IDPs, low immunization coverage, limited access to high-quality health care (trained staff and drugs) are likely to increase children's risk to illness and death, especially among rural populations.

PREVENTION AND CONTROL MEASURES

Case management	The priority is early recognition and adequate treatment of cases. The first-line antibiotic for cases aged under 5 years classified as pneumonia is co-trimoxazole; the second-line antibiotic is amoxicillin. Pre-referral antibiotics for severe cases that cannot tolerate oral antibiotics or for treatment of severe cases that cannot be referred are: – intramuscular chloramphenicol for children aged 2 months up to 5 years; and – intramuscular benzylpenicillin *and* gentamicin for infants aged under 2 months. Children with fever, in addition to cough or difficult breathing, may also be treated for malaria according to their exposure to malaria risk (high vs low malaria risk areas) and laboratory results (blood film) if these services are available. Supportive measures such as continued feeding to avoid malnutrition, vitamin A if indicated, antipyretics to reduce high fever and protection from cold (especially keeping young infants warm) are part of integrated case management. Prevention of low blood glucose is carried out for severe cases. Integrated management of illness is practised in any sick child seen by a provider trained in IMCI. Proper advice is given to caretakers of non-severe cases on home care, including compliance with antibiotic treatment instructions. Signs of malnutrition are assessed as this increases the risk of death due to pneumonia. Severely malnourished children (weight-for-height index <70%) must be referred to hospital. Source: *National guidelines on Integrated Management of Childhood Illness – IMCI* (revised in 2001).
Prevention	Health education on early danger signs for prompt care-seeking, good ventilation of housing and avoiding overcrowding. Adequate feeding, including exclusive breastfeeding, to avoid malnutrition. Improved immunization coverage.
Immunization	Measles, diphtheria and pertussis (whooping cough) immunization is effective in reducing the impact of ALRI. Immunization coverage rates for these antigens are currently suboptimal in Sudan.

2. AFRICAN TRYPANOSOMIASIS (AFRICAN SLEEPING SICKNESS)

DESCRIPTION

Infectious agent	Protozoan: *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense*
Case definition	**Clinical description** **1st stage (haemolymphatic involvement):** • A painful chancre (papular or nodular) at the primary site of tsetse fly bite (rare in *T.b. gambiense* infection). • Possibly fever, intense headache, insomnia, painless lymphadenopathy, anaemia, local oedema and rash. **2nd stage (neurological involvement):** • Parasites cross the blood–brain barrier and attack the central nervous system. • Cachexia, somnolence and signs of central nervous system involvement. – Possible protracted course of several years in *T.b. gambiense* infection. – Rapid and acute evolution in *T.b. rhodesiense* infection. – Both diseases are always fatal without treatment. **Laboratory tests available** • **Serological:** – Card Agglutination Trypanosomiasis Test (CATT): for *T.b. gambiense* only. – Immunofluorescent assay: for *T.b. rhodesiense* mainly; possibly for *T.b. gambiense.* • **Parasitological:** – Detection (microscopy) of trypanosomes in blood, lymph node aspirates or cerebrospinal fluid (CSF). **Case classification** • **Suspected*:** any case **without direct demonstration of the parasite** – that is compatible with the clinical description **and/or** – with a positive serology. • **Confirmed:** a case with direct demonstration of the parasite, compatible or not with the clinical description. – **1st stage:** parasite seen in blood and/or lymph nodes, with CSF containing no detectable trypanosomes and a leukocyte count <5/µl. – **2nd stage:** CSF containing trypanosomes and/or a leukocyte count >5/µl. * *In the 1st stage or early in the 2nd stage of the disease there are often no clinical signs or symptoms classically associated with the disease. Suspicion is then based on local risk of contracting the disease and local disease history.*
Mode of transmission	The disease is transmitted primarily by the bites from infected tsetse flies (*Glossina* spp.). Transmission is also possible through contamination with infected blood or through the placenta (congenital).
Incubation	– In *T.b. rhodesiense* infection: 3 days to a few weeks. – In *T.b. gambiense* infection: longer incubation period that can take several months or even years.

Period of communicability	The disease is communicable to the tsetse fly as long as the parasite is present in the blood of the infected person or animal (from 5 to 21 days after the infecting bite). Parasitaemia occurs in waves of varying intensity in untreated cases during all stages of the disease. Once infected, the tsetse fly remains infective for life (lifespan: 1–6 months).

EPIDEMIOLOGY

Burden	From January 2000 to November 2002, the figures collected from various and incomplete sources show that about 138 800 people were screened and about 6155 cases identified, of which about 48% were already in the neurological phase of the disease. The average prevalence rate calculated for these figures is 3.7%. The prevalence can reach more than 20% in some areas such as Ibba village in Kotobi, South Sudan. About 5 million people are at risk from African trypanosomiasis in Sudan, and 50 000 are estimated to be already infected. Given the focal nature of the disease, prevalence should refer only to the areas at risk. Aggregation of data at the national level is misleading and obscures the problem. It is almost impossible to measure incidence rates of *T.b. gambiense* sleeping sickness because the variable and long asymptomatic period of the disease make it impossible to predict with any accuracy when infection begins. Scant information on mortality exists outside hospital records, since most deaths occur in rural areas with poor or non-existing civil registration systems. Mortality in infants is particularly difficult to measure, even with systematic screening, because of the well known systematic underreporting of infant deaths. In addition, it is very difficult to obtain breakdowns by age or sex. A seroprevalence of 10–30% has been found in certain villages of southern Sudan.
Geographical distribution	Foci of *T.b. gambiense* are located in the southern part of Sudan, west of the Nile, within 100 km of the borders with Central African Republic, Democratic Republic of the Congo and Uganda. The main foci are Juba, Kajo Keji and Yei counties in Bahr Al Jebel State, and Maridi, Tambura, Yambio county in Western Equatoria State. Very little information is available on the current status of African trypanosomiasis in Bahr Al Ghazal and Eastern Equatoria states, but the area around Torit (Eastern Equatoria) is known to be heavily affected. Small foci of *T.b. rhodesiense* are located in southern Sudan on the east side of the Nile river, along the border with Ethiopia. An important feature of African trypanosomiasis is its focal nature. It tends to occur in circumscribed zones, and observed prevalence rates vary greatly from one geographical area to another, and even between villages within the same area.
Seasonality	The disease has no obvious seasonal pattern.
Recent epidemics in the country	Several major outbreaks have been observed periodically in southern Sudan since the early part of the 20th century. The first outbreak lasted from 1920 to 1929, the second from 1953 to 1961 and the third from 1975 to 1985. The 1975 epidemic primarily affected the Li Rangu, Yambio and N'zara areas. In 1977, Yambio district alone reported 614 new cases, all of which were self-reporting. After the resurgence of the disease in the late 1970s, a bilateral Sudanese–Belgian Sleeping Sickness Control Programme limited incidence of the disease, which had been virtually eliminated by 1983. However, control activities collapsed in 1990 when fighting in the civil war intensified. The disease has gained epidemic proportions since the mid-1990s; today, Sudan is included among the four worst-hit countries (with Angola, Democratic Republic of the Congo and Uganda), where African trypanosomiasis is epidemic due to high prevalence and an important transmission level.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Risk of settlement in a high-transmission area. Uncultivated land often becomes resettlement area for displaced populations.
Overcrowding	**No**	Tsetse density is not related to the density of the human population.
Poor access to health services	**Yes**	The complex nature of the disease requires efficient health structures and trained personnel for diagnosis and treatment.
Food shortages	**No**	
Lack of safe water and poor sanitation	**No**	The tsetse fly is not attracted by dirty water.
Others	**Yes**	It is a neglected disease.
Risk assessment conclusions		Southern Sudan is experiencing a resurgence of epidemic sleeping sickness: the transmission rate and prevalence are increasing rapidly. War has exacerbated the breakdown of surveillance, case detection and treatment. Access to populations in epidemic areas has so far been extremely difficult. The health infrastructure and services capacity has almost collapsed. The number of people living in areas at risk for sleeping sickness in southern Sudan can be estimated at 1–2 million, but reliable data are not available. Prevalence of confirmed *T.b. gambiense* infection in humans now exceeds 5% in several foci.

PREVENTION AND CONTROL MEASURES

Case management	Early screening and diagnosis are essential, as treatment is easier in the 1st stage of the disease (fewer injections required, no psychiatric symptoms, has lower risk and can be administered on an outpatient basis. Diagnosis and treatment require trained personnel; self-treatment is not possible. All confirmed cases must be treated immediately. Most available drugs are old, difficult to administer in suboptimal conditions and frequently unsuccessful. ***T.b. gambiense* infection:** • 1st stage: – **Pentamidine** (4 mg/kg per day) IM for 7 consecutive days on an outpatient basis. • 2nd stage: – **Melarsoprol.** Hospitalization with three series of injections administered with a rest period of 8–10 days between each series. A series consists of one daily injection of 3.6 mg/kg melarsoprol IV for 3 consecutive days. – If melarsoprol treatment failure occurs, use **eflornithine** 400 mg/kg per day administered in four daily slow infusions (lasting approximately 2 hours). Infusions are given every 6 hours, representing a dose of 100 mg/kg per infusion. ***T.b. rhodesiense* infection:** • 1st stage: – **Suramin.** The recommended dosage is 20 mg/kg per day with a maximum dose of 1 g per injection. The drug is administered intravenously at the rate of one weekly injection. The treatment course is 5 weeks for a total of five injections. • 2nd stage: – **Melarsoprol.** Hospitalization with three series of injections administered with a rest period of 8–10 days between each series. A series consists of one daily injection of 3.6 mg/kg melarsoprol IV for 3 consecutive days. ***Note:*** *Melarsoprol causes reactive encephalopathy in 5–10% of patients, with fatal outcome in about 50% of cases. The treatment has a 10–30% failure rate, probably due to pharmacological resistance. Increasing rates of resistance to melarsoprol (as high as 25%) have been reported from various countries.* A Human African Trypanosomiasis Treatment and Drug Resistance Network has been established by WHO. Four working groups deal with: (a) Drug availability and accessibility; (b) Coordination of drug development and clinical trials; (c) Research on resistance and treatment schedules; and (d) Surveillance of resistance. **Procurement of drugs** Since 2001, a public – private partnership signed by WHO has made all drugs widely available. The drugs are donated to WHO. Requests for supplies are made to WHO by governments of disease-endemic countries and organizations working in association with these governments. Stock control and delivery of the drugs are undertaken by *Médecins sans Frontières* in accordance with WHO instructions. All the drugs are provided free of charge: recipient countries pay only for transport costs and customs charges.

Prevention	• **Routine preventive measures** through public education on the following should be encouraged: – Avoidance of known foci of sleeping sickness and/or tsetse infestation – Wearing suitable clothing (including long sleeves and long trousers) in endemic areas – Routine use of insect repellents and mosquito nets. • **Case detection** through containment of the human reservoirs through periodical population screening and chemotherapy of cases remains the cornerstone of disease control for *gambiense* sleeping sickness. Active periodical screening (active case-finding) of the population of endemic foci by mobile screening teams is the best option, since infected subjects can remain asymptomatic and contagious for months or years before developing overt symptomatology. Screening usually comprises CATT testing of the entire population visited by teams. Because *rhodesiense* sleeping sickness is an acute disease, passive case-finding by fixed posts is more appropriate, since symptoms are severe and patients will tend to seek health care voluntarily. • **Vector control** through tsetse fly control programmes: – Application of residual insecticides or aerosol insecticides – Use of insecticide-impregnated traps and screens – Destruction of tsetse habitats by selective clearing of the vegetation: clearing bushes and tall grasses around villages is useful when peridomestic transmission occurs. Indiscriminate destruction of vegetation is NOT recommended. Since 1997, a community-based vector trapping project has been implemented in Tambura county (Western Equatoria): more than 3000 pyramidal traps made locally and maintained by volunteers have been placed at sites where people are likely to come into contact with tsetse flies. Between 1997 and 1999, the seroprevalence of African trypanosomiasis in Tambura county villages in which screening, drug treatment and vector control activities were being conducted dropped from almost 9% to less than 2%. • **Prohibition of blood donation** from those who live (or have stayed) in endemic areas. The Government of Sudan has established a National Committee for Tsetse and Trypanosomiasis Control (NCTTC) to enhance human trypanosomiasis management activities and to effectively mobilize and manage resources allocated for the control or eradication of the disease from Sudan. The NCTTC includes the Federal Ministry of Health, the Bahr Al Jebel Regional Ministry of Health, the Tropical Medicine Research Institute and the Central Veterinary Research Laboratories. These institutions are involved in surveillance and case detection activities, hospitalization of cases, drug resistance monitoring, training of sleeping sickness staff, vector surveys and studies on the animal reservoir. Unfortunately, control activities are currently hampered by lack of adequate funding.
Epidemic control	Mass surveys to identify affected areas. Early identification of infection in the community, followed by treatment. Urgent implementation of tsetse fly control measures (e.g. aerosol insecticides sprayed by helicopter and fixed-wing aircraft).

3. BACILLARY DYSENTRY (SHIGELLOSIS)

DESCRIPTION

Infectious agent	Bacterium: genus *Shigella,* of which *Shigella dysenteriae* type 1 (Sd1) causes the most severe disease and is the only strain responsible for epidemics.
Case definition	**Case classification** **Suspected:** Diarrhoea with visible blood in the stools. **Confirmed:** A case corresponding to the clinical case definition with isolation of *Shigella* from stools.
Mode of transmission	Faecal–oral route, particularly through contaminated water and food.
Incubation	Incubation period is usually 1–3 days; may be up to 1 week for *S. dysenteriae* type 1.
Period of communicability	During acute infection and until 4 weeks after illness (without treatment). With appropriate treatment 2–3 days. Asymptomatic carriers exist.

EPIDEMIOLOGY

Burden	Although many suspected cases exist, most cases have not been confirmed.
Geographical distribution	Diffuse distribution with no foci.
Seasonality	Cases occur throughout the year. Seasonal incidence patterns are not constant over years.
Alert threshold	Five or more linked cases must be investigated further.
Recent epidemics in the country	**2004 March–June***.* An average of 50 cases per week was reported in Darfur. Sd1 was confirmed by laboratory. In Week 25, 100 cases and 2 deaths were reported*.* **2001 February***.* 7 deaths were reported from Acumcum (Western Bahr Al Ghazal). Many cases were also reported but figures were not available. *S.* dysenteriae type 1 (Sd 1*)* was isolated from stool samples. **1999 March–April***.* During an outbreak of relapsing fever in Rumbek county (Lakes State), cases of bloody diarrhoea were observed and confirmed as shigellosis.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Spread of the infectious agent.
Overcrowding	**Yes**	Very important for transmission of the disease.
Poor access to health services	**Yes**	Early detection and containment of cases are paramount in reducing transmission. Without proper treatment, the case-fatality rate of *S. dysenteriae* type 1 can be as high as 10% in children aged under 10 years.
Food shortages	**No**	However, malnutrition increases gastrointestinal tract susceptibility to invasiveness of the organism and severity of disease.
Lack of safe water and poor sanitation	**Yes**	The most important risk factor.

Others	**No**	Contaminated food, lack of soap and poor hygiene are also very important risk factors.
Risk assessment conclusions	Overcrowding, lack of safe water, and inadequate sanitation promote the risk of infection. The risk of epidemics of *S. dysenteriae* type 1 is high in camp settings (up to one-third of the population at risk may be affected). Early detection of cases and institution of antibiotic therapy is essential.	

PREVENTION AND CONTROL MEASURES

Case management	Early and appropriate therapy is very important: treatment with an effective antimicrobial can reduce the severity and duration of shigellosis. Selection depends on resistance patterns of the bacteria and drug availability. The problem of rapid acquisition of antimicrobial resistance in treating Shigella dysentery in Africa is a cause of concern. It is therefore important to confirm the sensitivity of *S. dysenteriae* to antibiotics in the early stages of an outbreak of shigellosis. Resistance patterns may vary during the course of an outbreak and regaular stool sampling is required. Ciprofloxacin is the current first-line antibiotic of choice recommended for treatment of *S .dysenteriae* type 1. Supportive treatment using oral rehydration salts (ORS), continued feeding (frequent small meals) and antipyretics to reduce high fever is also essential. *S. dysenteriae* type 1 is often more severe or fatal in young children, the elderly and the malnourished, and prompt treatment with antibiotics is essential. If in short supply, antibiotics should be reserved for such high-risk groups. See: Annex 6 of this Toolkit - *Case Management of epidemic-prone diseases.*
Epidemic control	Inform the health authorities when one or more suspected cases are identified. Early detection and notification of epidemic dysentery, especially among adults, enables timely mobilization of resources for appropriate Case management and control. Confirm the outbreak in accordance with WHO guidelines. See: Annex 6 of this Toolkit - *Case Management of epidemic-prone diseases.* Rectal swabs from suspected cases should be collected and shipped refrigerated to laboratories in an appropriate medium (e.g. Cary-Blair medium) for culture to confirm the diagnosis of Sd1. It is recommended that at least 10 cases be used to confirm the cause, identify antibiotic sensitivity and verify the outbreak. Once confirmed, it is not necessary to obtain laboratory confirmation for every patient. Testing of Sd1 isolates for antimicrobial sensitivity should be done at regular intervals to determine whether treatment guidelines remain appropriate. International referral laboratories are available to assist in identification of the organism and confirmation of the antimicrobial resistance pattern. **Do not wait for laboratory results before starting treatment/control activities.**
Prevention	**See:** – *Diarrhoeal diseases (others)* and Appendix 3: *Safe water and sanitation* in this Profile. – *Guidelines for the control of epidemics due to Shigella dysenteriae type 1.* Geneva, WHO, 1995 (WHO/CDR/95.4 available at: http://www.who.int/emc-documents/cholera/whocdr954c.html).

4. CHOLERA

DESCRIPTION

Infectious agent	Bacterium: *Vibrio cholerae*
Case definition	**A cholera outbreak should be suspected if:** A person aged older than 5 years develops severe dehydration or dies from acute watery diarrhoea (clinical case definition); **or** There is a sudden increase in the daily number of patients with acute watery diarrhoea, especially patients who pass the "rice water" stools typical of cholera. **Confirmed case:** Isolation of *Vibrio cholerae* O1 or O139 from stools in any patient with diarrhoea.
Mode of transmission	**Faecal – oral route** **1. Person-to-person transmission** – when taking care of cholera patients. – through direct contact with the bodies of deceased cholera patients (e.g. washing and preparing the body for funeral ceremonies). **2. Drinking contaminated water** **3. Eating food (fruits and vegetables) contaminated through** – water – soil – contamination *during* preparation (rice, millet, food from street vendors) – contaminated seafood. **4. Indirect contamination (hands)**
Incubation	Incubation period is usually a few hours to 5 days.
Period of communicability	During the symptomatic phase until 2–3 days after recovery; very rarely for months. Asymptomatic carriers are common.

EPIDEMIOLOGY

Burden	Although no official data are available, cases of cholera are known to occur in the country.
Geographical distribution	There is no definite geographical distribution of the disease.
Seasonality	All of the outbreaks mentioned below occurred between March and June.
Alert threshold	Any suspected case must be investigated.

Recent epidemics in the country	No official data are available. **2002 February–April**. As of 18 April, 109 cases of "acute watery diarrhoea" including 1 death had been reported from Kerker in the Nuba mountains area of Southern Kordofan State. All cases were children aged under 5 years. **2001 April–May**. A total of 65 cases of "acute watery diarrhoea" including 5 deaths were reported from Wudier and Beih (Upper Nile). (Source: WHO SS Health Update) **1999** – Since early March, Padak, Mading, Wanding, Lankien, Akobo and Burmat areas have reported a total of 892 cases of "acute watery diarrhoea" with 24 deaths up to 27 April. The outbreak affected mainly Jongli State in areas south of river Sobat. (Source: WHO) **1996** – In April, an outbreak of cholera and severe diarrhoeal diseases spread rapidly through rebel-held areas in southern Sudan, with more than 12 000 cases reported in 6 weeks. The outbreak resulted in at least 1800 deaths. Although the exact numbers are unknown, because several locations reporting outbreaks were inaccessible, case-fatality rates were extremely high in those locations where data could be confirmed. (Source: United Nations) **1985 May–June**. 1175 cases of cholera with 41 presumed home deaths and 13 inpatient deaths were registered among displaced populations from Ethiopia settled in two adjacent camps near Khashm el Girba in eastern Sudan (Kassala State).

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Spread of the infectious agent.
Overcrowding	**Yes**	Very important.
Poor access to health services	**Yes**	Early detection and containment of cases are paramount in reducing transmission.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	The most important risk factor.
Others	**No**	
Risk assessment conclusions		The high prevalence of acute and chronic malnutrition could also lead to increased susceptibility to severe disease. Cholera can result in severe dehydration within a few hours. The case fatality rate may surpass 50% in those presenting with severe dehyadration if untreated. With good case management case fatality rate should be below 1%. Risk remains high while there is inadequate water and sanitation, population displacement and overcrowding. Without adequate access to appropriate health care, case fatality is very high.

PREVENTION AND CONTROL MEASURES

Case management	The mainstay of case management for cholera is the treatment of dehydration using ORS or IV fluids (Ringer's lactate). Use of antibiotics (doxycycline/tetracycline) is not essential for disease treatment but may be used to reduce the volume of diarrhoea (and of the rehydration solutions required), shorten its duration and the period of vibrio excretion. Antimicrobial sensitivity patterns should be assessed in order to select the appropriate antibiotic. The case-fatality rate can be extremely high (from 5% up to 40%) without proper treatment. With appropriate case management, it is less than 1%.
Epidemic control	Inform the health authorities immediately if one or more suspected cases are identified. Confirm the outbreak in accordance with WHO guidelines. Stool samples must be taken with a rectal swab and transported in Cary-Blair medium. If a transport medium is not available, a cotton-tipped rectal swab can be soaked in the liquid stool, placed in a sterile plastic bag, tightly sealed and sent to the laboratory. It is recommended that at least 10 cases be used to confirm the cause, identify antibiotic sensitivity and verify the outbreak. Once confirmed, it is not necessary to obtain laboratory confirmation for every patient. **Do not wait for laboratory results before starting treatment/control activities:** – Ensure prompt treatment and confirm the diagnosis – Isolate cases in cholera treatment centres – Provide adequate health education – Ensure access to safe water and proper sanitation.
Prevention	**See:** "Prevention" in *Diarrhoeal diseases (others)* and Appendix 3: *Safe water and sanitation* in this Communicable Disease Profile.
Immunization	The use of oral cholera vaccine (OCV) is considered an additional public health tool to recommended cholera control measures such as provision of safe water and adequate sanitation. OCV is recommended for populations to limit the risk of : - occurrence of cholera outbreaks in endemic areas. - spread and incidence of cholera during an outbreak. Two oral vaccines are currently available: the killed cholera vaccine (WC/rBS; 2 doses) and the attenuated live vaccine (CVD103-HgR; single dose). Both vaccines have been licensed in some countries. **Use of the single dose OCV is possible once an outbreak has started. The two dose OCV cannot be used once an outbreak has started (**See: Joint WHO-UNICEF statement fro Cholera vaccine use in tsunami affected areas. http://www.who.int/cholera/tsunami_choleravaccine/en/index.html) For more specific information on cholera vaccines and their use, contact the Global Task Force on Cholera Control at WHO Geneva: cholera@who.int

References	**See:** – Leaflet, *First steps for managing an outbreak of acute diarrhoea.* Geneva, WHO, 2003 (WHO/CDS/CSR/NCS/2003.7 available at www.who.int/csr/diseases/cholera). – *Guidelines for collection of specimens for laboratory testing* in this Toolkit (Document 7). – *Cholera Outbreak: Assessing the outbreak response and improving preparedness.* WHO/CDS/CPE/ZFK/2004.4 – *Cholera vaccines: a new public health tool? Report, WHO meeting, 10–11 December 2002, Geneva, Switzerland*. Geneva, WHO, 2004 (WHO/CDS/CPE/ZFK/2004.5).

5. DIARRHOEAL DISEASES (OTHERS)

DESCRIPTION

Infectious agent	**Bacteria:** such as *Salmonellae* (commonly S. *enteritidis, S. typhimurium)* and *Escherichia coli.* The bacteria that cause the most severe outbreaks are *Shigella dysenteriae* type 1 and *Vibrio cholerae (*see *Bacillary dysentery* and *Cholera).* **Protozoa:** such as *Entamoeba histolytica, Giardia lamblia* and *Cryptosporidium parvum.* **Viruses:** such as Rotavirus and Norwalk virus.
Case definition	**Clinical case definition** Three or more abnormally loose or fluid stools over a period of 24 hours.
Mode of transmission	Faecal–oral route, particularly through contaminated water and food.
Incubation	*Salmonella* generally requires an 8–48 hour incubation period, whereas that for *E. coli* is typically longer at 2–8 days (median of 3–4 days); both usually last between 2–5 days. The average incubation period is 2–4 weeks for *E. histolytica*, 7–10 days for *G. lamblia* and 7 days for *C. parvum*. The incubation period for *Rotavirus is* about 48 hours, and symptoms may last for up to 1 week.
Period of communicability	During the acute stage of the disease and for the duration of faecal excretion. Temporary *Salmonella* carriers can continue to exist for several months.

EPIDEMIOLOGY

Burden	**Year** **Number of cases reported nationally** 2000 32 48423 2001 27 09955 2002 10 66893 (Data source: WHO Sudan Country Office, 2004)
Geographical distribution	Throughout the country.
Seasonality	Diarrhoeal disease rates are higher in summer than in winter.
Alert threshold	An increase in the number of cases above what is expected compared with previous years.
Recent epidemics in the country	1999. An outbreak in Maywut (Upper Nile) caused 65 cases and one death. A non-typhoid *Salmonella* was found to be the responsible microorganism.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Can import cases.
Overcrowding	**Yes**	Very important.
Poor access to health services	**Yes**	Early detection and containment of the cases are paramount in reducing transmission.

Food shortages	**No**	However, malnutrition increases gastrointestinal tract susceptibility to invasiveness of the organism and severity of disease.
Lack of safe water and poor sanitation	**Yes**	The most important risk factor: prevention of diarrhoeal diseases depends on the provision and use of safe water, adequate sanitation and health education. The supply of adequate quantities of water should be one of the highest priorities for camp planners. The emergency requirement is 20 litres/person per day. Common sources of infection in emergency situations are: – Contaminated water sources (e.g. by faecally-contaminated surface water entering an incompletely sealed well) or during storage (e.g. by contact with hands soiled by faeces). – Shared water containers and cooking pots.
Others	**Yes**	Poor hygiene, lack of soap, contaminated food items.
Risk assessment conclusions		In camp situations, diarrhoeal diseases can account for 25–40% of deaths in the acute phase of an emergency. More than 80% of deaths usually occur in children aged under 2 years.

PREVENTION AND CONTROL MEASURES

Case management	• Prevention – using home made fluids and ORS – and treatment of dehydration – with ORS or IV fluids (Ringer's lactate) for severely dehydrated patients – is the mainstay of case management of diarrhoeal illness, together with continuing feeding especially in children. – Reduction of mortality due to diarrhoeal diseases is primarily related to effective management of dehydration particularly in children. • Use of antibiotics is dependent on the infectious agent. • Resume feeding with a normal diet when vomiting has stopped. It is important to separate those who are eating from those who are not. Food should be cooked on site. Continue breastfeeding infants and young children.
Epidemic control	• Inform the health authorities immediately if an increase in the number of cases above what is expected is identified. • Confirm the diagnosis and ensure prompt treatment. • Confirm the outbreak in accordance with WHO guidelines.

Prevention	**Safe drinking-water** Provision of an adequate supply, collection and storage system. Provision of information on the importance of clean water, also covering system maintenance and household storage. (See Appendix 3*: Safe water and sanitation*). **Safe disposal of human excreta** Provision of adequate facilities for the disposal of human waste. Provision of information on the importance of human waste disposal, also covering use and maintenance of facilities. **Food safety** Provision of adequate storage facilities for food (both uncooked and cooked), cooking utensils, adequate quantity of water and fuel to allow for cooking and reheating. Health education on the importance of food safety and safe food handling. **Hand-washing with soap** Provision of soap in sufficient quantities for hand-washing, bathing and laundry. Health education on the relationship between disease spread and lack of or poor hand-washing practices. Demonstration on the importance of thorough hand-washing. **Breastfeeding** Provision of information on the protective qualities of breastfeeding and the importance of breastfeeding ill children. Practical support for breastfeeding ill children.

6. DIPHTHERIA

DESCRIPTION

Infectious agent	Bacterium: *Corynebacterium diphtheriae*
Case definition	**Clinical description** Upper respiratory tract illness with laryngitis or pharyngitis or tonsillitis **plus** adherent membranes of tonsils or nasopharynx. **Laboratory confirmation:** isolation of C. *diphtheriae* from a clinical specimen. **Case classification** **Suspected case:** not applicable. **Probable case:** a case that meets the clinical description. **Confirmed case:** a probable case confirmed by laboratory or epidemiologically linked to a laboratory-confirmed case. **Carrier:** presence of C. *diphtheriae* in nasopharynx, no symptoms. ***Note**: Persons with positive* C. diphtheriae *identification who do not meet the clinical description (e.g. asymptomatic carriers) should not be reported as probable or confirmed cases.*
Mode of transmission	Contact (usually direct, rarely indirect) with the respiratory droplets of a case or carrier. In rare cases, the disease may be transmitted through foodstuffs (raw milk has served as a vehicle).
Incubation	Usually 2–5 days; occasionally longer.
Period of communicability	Until virulent bacilli have disappeared from discharges and lesions; usually 2 weeks or less and seldom more than 4 weeks. The rare chronic carrier can shed bacilli for 6 months or more. The disease is usually not contagious 48 hours after antibiotics are instituted.

EPIDEMIOLOGY

Burden	**Number of cases reported nationally** 2003: 156 cases 2002: 26 cases 2001: 28 cases 2000: 26 cases 1999: 21 cases 1998: 67 cases 1997: 15 cases 1990: 1342 cases 1980: 587 cases (Data source: WHO/IVB data, 2004)
Geographical distribution	Throughout the country.
Seasonality	Seasonal incidence patterns are not constant over years.
Alert threshold	Once suspected, a probable or confirmed case must be investigated.
Recent epidemics	No outbreaks have been reported recently.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Importation.
Overcrowding	**Yes**	Crowded conditions facilitate transmission.
Poor access to health services	**Yes**	No access to routine immunization services. Early detection and containment of cases are paramount to reduce transmission.
Food shortages	**No**	
Lack of safe water and poor sanitation	**No**	
Others	**Yes**	Low DPT3 coverage (<80%). **DTP3 coverage** **2001:** 71% (46% by WHO–UNICEF estimates) **2000:** 65% **1999:** 79% **1998:** 70% **1997:** 79% **1990:** 62% **1980:** 1% (Data source: WHO/Sudan country estimates)
Risk assessment conclusions		Given that DPT3 coverage is below the recommended standard, outbreaks can be expected. Detection of outbreaks may be hampered due to poor access to health centres and poorly trained personnel. Additionally, outbreaks occur when social or natural conditions lead to overcrowding of susceptible groups, especially infants and children. This frequently occurs when there are large-scale movements of non-immunized populations.

PREVENTION AND CONTROL MEASURES

Introduction	The control of diphtheria is based on three measures: – Ensuring high population immunity through vaccination (primary prevention). – Rapid investigation and treatment of contacts (secondary prevention of spread). – Early diagnosis and proper case management (tertiary prevention of complications and deaths).
Immunization	Immunize the population at risk as soon as possible. In an epidemic involving adults, immunize groups that are most affected and at highest risk. Repeat immunization procedures 1 month later to provide at least 2 doses to recipients. Diphtheria–toxoid-containing vaccine (preferably the adult form of tetanus toxoid with reduced amount of diphtheria toxoid – Td) should be given. To ensure injection safety during immunization, auto-disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.

Case management	Diphtheria antitoxin and antibiotic therapy are the cornerstones of therapy for diphtheria. The antibodies neutralize toxin only before its entry into cells, and it is therefore critical that diphtheria antitoxin be administered s soon as a presumptive diagnosis has been made. • Antibiotic therapy, by killing the organism, has three benefits: – Termination of toxin production – Improvement of local infection – Prevention of spread of the organism to uninfected persons. **Do not wait for laboratory results before starting treatment/control activities.** **<u>Patients</u>** Diphtheria antitoxin IM (20 000–100 000 units) in a single dose, immediately after throat swabs have been taken **plus** Procaine penicillin IM (25 000–50 000 units/kg per day for children; 1.2 million units/kg per day for adults in 2 divided doses), **or** parenteral erythromycin (40–50 mg/kg per day with a maximum of 2 g per day) until the patient can swallow **then** Oral phenoxymethylpenicillin (125–250 mg) in 4 doses per day, **or oral** erythromycin (40–50 mg/kg per day with a maximum of 2 g per day) in 4 divided doses. ***Antibiotic treatment should be continued for a total period of 14 days.*** Isolation: strict isolation for pharyngeal diphtheria, or contact isolation only for cutaneous diphtheria for a total of 14 days. **<u>Close contacts</u>**[1] Surveillance for 7 days for close contacts, regardless of vaccination status, and throat cultures. All close contacts must receive a single dose of benzathine benzylpenicillin G IM (600 000 units for children aged under 6 years; 1.2 million units for those aged 6 years or older). Erythromycin can be used also as second choice. If culture is positive, give antibiotics as for patients above. **<u>Carriers</u>** All carriers must receive a single dose of benzathine benzylpenicillin G IM (600 000 units for children aged under 6 years; 1.2 million units for those aged 6 years and older). ***Note:*** *Clinical diphtheria does not necessarily confer natural immunity, and patients should therefore be vaccinated before discharge from a health facility.*

[1] Close contacts include household members and other persons with a history of direct contact with a case, as well as health care workers exposed to oral or respiratory secretions of a case.

Epidemic control	Inform the health authorities when one or more suspected cases are identified. Confirm the suspected outbreak, following WHO guidelines. Investigate any probable case: check whether it fulfils the case definition, record date of onset, age and vaccination status. Investigate any probable case: check whether it fulfils the case definition, record date of onset, age and vaccination status. Confirm the diagnosis: collect both nasal and pharyngeal swabs for culture and swabs from any wounds or skin lesions. If appropriate facilities are available, determine the biotype and toxigenicity of C. *diphtheriae.* Identify close contacts and define population groups at high risk. Adult contacts must avoid contact with children and must not be allowed to undertake food handling until proven not to be carriers. Implement outbreak response measures. Give priority to case management and immunization of population in areas not yet affected where the outbreak is likely to spread. Immunize the population at risk as soon as possible, especially children. In an epidemic involving adults, immunize groups that are most affected and at highest risk. Repeat immunization procedures 1 month later to provide at least 2 doses to recipients. In endemic situations, Td vaccine (a combination of diphtheria and tetanus toxoids with reduced diphtheria content) should preferably be given. To ensure safety of injection during immunization, auto-disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.

7. DRACUNCULIASIS (GUINEA-WORM DISEASE)

DESCRIPTION

Infectious agent	Nematode: *Dracunculus medinensis*
Case definition	**Clinical description:** Diagnosis is usually easy and unambiguous: the gravid female worm (up to 1 m long) emerges through the skin of any part of the body about 1 year after infection. When the anterior part of the worm reaches the surface of the skin, an intensely painful oedema and a papule are formed. The papule is succeeded (within 1–3 days) by a blister that ruptures (after 3–5 days) leaving a small superficial ulcer. Systemic symptoms include fever, nausea and vomiting. Functional lesions of the affected limb are frequent. Lower extremities are involved in 90% of cases, with resulting crippling. Secondary bacterial infections are also of major concern. No immunity to infection develops, and people in endemic areas suffer from infection year after year. Each infection lasts about 1 year. **Case definition:** Anyone exhibiting or having a history of a skin lesion with the emergence of a guinea worm within the current year.
Mode of transmission	Swallowing of water containing minute crustacean copepods *(Cyclops* or "water fleas", which measure 1–2 mm) that have ingested larvae of *D. medinensis* discharged by the adult female worm into stagnant water bodies. There is no known animal reservoir of the infection.
Incubation	The female gravid worm emerges through the skin (most frequently of the legs) about 12 months after larvae have been introduced into the human body.
Period of communicability	12–50 days after rupture of vesicle. This results from the sum of the following periods: – 2–3 weeks: the period from rupture of vesicle until larvae have been completely evacuated from the uterus of the gravid worm. – About 5 days: the period during which larvae are infective for the copepods in water. – 12–14 days to about 3 weeks after ingestion by copepods: the period during which the larvae become infective for humans (at temperatures exceeding 25 °C).

EPIDEMIOLOGY

Burden	**2001.** Sudan reported 49 471 cases, equivalent to 78.0% of all cases reported worldwide (63 717). **2000.** Sudan reported 54 890 cases, equivalent to 72.9% of all cases reported worldwide (75 223). **1999.** Sudan reported 66 097 cases, equivalent to 68.6% of all cases reported worldwide (96 293). **1998.** Sudan reported 47 977 cases, equivalent to 61.0% of all cases reported worldwide (78 557). **1997.** Sudan reported 43 596 cases, equivalent to of 55.9% of all cases reported worldwide (77 863) ***Note**: Dracunculiasis transmission has been confined to Africa since 1998. (The last indigenous cases outside Africa were reported from Yemen in September 1997.)*

Geographical distribution	Almost all of Sudan's cases come from southern states. Moreover, southern Sudan is a concern for neighbouring areas, since it exports cases to other Sudanese states and abroad: 28, 175, 7, 16 and 32 cases have been exported annually to adjacent countries (Ethiopia, Uganda, Kenya and the Central African Republican) in 1997–2001. Within Sudan, the northern states have already almost interrupted transmission of dracunculiasis. Only 85 indigenous cases were reported from 7 of the 16 northern states in 2001, compared with 4053 cases from the northern states in 1995.
Seasonality	The disease is seasonal, occurring with patterns depending on climatic factors, especially rainfall. In the Sahelian zone, transmission generally occurs in the rainy season (May–August) when surface water is available. In the humid savannah zone, the peak transmission period usually occurs in the dry season (November–January) when drinking-water sources are most scarce and heavily contaminated. In Sudan, the majority of cases are reported during the rainy season between May and October when the worm emerges after its 12-month incubation period. This is also the period during which most infections occur.
Recent epidemics in the country	Dracunculiasis is an endemic disease, with little likelihood of rapid changes in incidence. However, in hyperendemic situations, field surveys can be performed to determine prevalence of infection, discover high-risk sources of water and apply control measures (see below).

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Infected individuals can "export" the disease to non-endemic areas provided the disease cycle can complete itself.
Overcrowding	**Yes**	Overcrowding can lead more people to share the same body of water where *D. medinensis* larvae have been discharged.
Poor access to health services	**Yes**	The difficulties of implementing any public health programme in Sudan due to the civil conflict are responsible for high disease transmission in the country.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	Among the most important factors. Insufficient water and poor hygiene practices which lead to people fetch and washing themselves in the same body of water.
Others	**No**	

Risk assessment conclusions	Sudan reported 78% of all dracunculiasis cases reported globally in 2001. Almost all of Sudan's cases are in the southern states, where the civil war has limited for a long time accessibility to endemic areas. The country's proportion of global dracunculiasis cases has steadily increased during the past 7 years as the number of cases is reduced in all other endemic countries. The number of reported cases has decreased during the past 3 years despite intensive campaigns to reduce under-reporting. However, this decrease is not considered wholly representative because many endemic villages in the south are inaccessible as a result of civil disturbance. Although rarely fatal, dracunculiasis is of great socioeconomic importance. Persons with this disease are incapacitated as a result of pain caused by the primary wound at the exit point of the worms and by associated secondary infections. Temporary disability usually lasts for periods averaging almost 3 months (usually 10–11 weeks), mainly because: • several worms can be expelled successively, • migration and emergence of the worms occur in sensitive parts of the body, e.g. the sole of the feet, • serious secondary bacterial infection frequently sets in subsequent to the accidental rupture of the worm. The emerging of the worm often happens at the busiest time of the year when people need to plant or harvest their crops, and half or more of a village population may be affected simultaneously. In addition to its impact on agricultural productivity, dracunculiasis is also a major cause of absenteeism from school. Moreover, it has been observed that, when disabled adult members of a household are prevented from fully performing their agricultural or domestic activities as a result of dracunculiasis, the nutritional status of children in the same household will deteriorate in the following year due to both lack of food and negligence in the care of children. Man-made water-catchment ponds such as *haffir*, shallow wells and ponds are the main source of transmission in Sudan, and the epidemiology of the disease is determined largely by the use of these open water sources. Sudan currently has the highest disease burden of dracunculiasis in the world. Vigilant surveillance and appropriate public health interventions are key for the global effort to eliminate the disease.

PREVENTION AND CONTROL MEASURES

Case management	No drugs are currently available to kill the adult worm. Slow extraction of the emergent guinea worm, with appropriate antibiotic cover, is the most effective measure. Once a worm emerges, use a matchstick to roll it out gently a few centimetres a day until the worm has been removed. Never break the worm, and never pull it. Bandage the wound after applying an antibiotic ointment to prevent superinfection of the lesion; 24 hours later, remove the bandages and roll the part of the worm that has emerged. Repeat this procedure until the whole worm has been removed (usually 10–20 days). Care must be taken to ensure that the worm does not break during the course of extraction. Although practised in certain endemic countries, surgical extraction is NOT recommended.
Prevention	**Provision of safe water sources:** this is the most expensive, and the most durable, intervention. It also has the advantage of providing other important benefits besides eliminating the guinea worm. **Control of copepod populations** in ponds, tanks, reservoirs and step wells. — Insecticide of choice for stagnant sources of water: temephos (Abate®), which is effective and safe. — Formulation and dosage: based on the estimated amount of water present. — Time of application: the insecticide must be applied regularly, with a maximum interval of 28 days between applications to ponds less than 500 m in diameter during transmission season of known endemic villages and in villages newly reporting cases. The application is most effective after a flood has receded. **Health education:** programmes should be focused on the following two messages: 1. Villagers with blisters or ulcers should not enter any source of drinking-water. It is well known that infected persons try to relieve the burning sensation by immersing the affected part of the body in local water sources. This should be discouraged. 2. Guinea-worm infection comes from drinking-water. Water should therefore be: – boiled (this is usually impractical given the scarcity or high cost of wood or other fuel); or – chlorinated; or – filtered to remove copepods. Systematic filtering of drinking-water derived from ponds, shallow unprotected wells or from surface water should be encouraged: use of finely-meshed cloth filter, straw filter, or, preferably a filter made from a 0.15 mm nylon mesh, is the recommended option.
Immunization	No vaccine is available. However, populations at high risk should be immunized against tetanus.

8. EBOLA HAEMORRHAGIC FEVER

DESCRIPTION

Infectious agent	Ebola virus, belonging to the genus *Filovirus*.
Case definition	**Clinical description** Presentation may be very nonspecific. Initial symptoms include acute fever, diarrhoea that can be bloody (referred to as *diarrhee rouge* in francophone Africa) and vomiting. Headache, nausea and abdominal pain are common. Conjunctival injection, dysphagia and haemorrhagic symptoms (nosebleeds, bleeding gums, vomiting of blood, blood in stools, purpura) may further develop. Some patients may show a maculopapular rash on the trunk. Dehydration and significant wasting occur as the disease progresses. At a later stage, frequent involvement of the central nervous system occurs, manifested by somnolence, delirium or coma. The case-fatality rate ranges from 50% to 90%. **Laboratory criteria:** **Confirmation** – Positive ELISA antigen detection or IgM capture, or – Positive virus isolation (only in a laboratory of Biosafety Level 4), **or** – Positive skin biopsy (immunohistochemistry), **or** – Positive PCR with sequence confirmation. **Case classification*:** **Suspected:** a case that is compatible with the clinical description. **Probable (in epidemic situation):** – Any person having had contact with a clinical case and presenting with acute fever, **or** – Any person presenting with acute fever and three of the following symptoms: headache, vomiting/nausea, loss of appetite, diarrhoea, intense fatigue, abdominal pain, general or articular pain, difficulty in swallowing, difficulty in breathing, hiccups, **or** – Any unexplained death. **Confirmed:** Any suspected or probable case that is laboratory-confirmed. **Contact (in epidemic situation):** An asymptomatic person having had physical contact within the past 21 days with a confirmed or probable case or his/her body fluids (e.g. care for patient, participation in a burial ceremony, handling of potentially infected laboratory specimens). ** Case classification should be tailored according to circumstances locally identified in the field (e.g. including contact with sick animals or animals with abnormal behaviour).*
Mode of transmission	Person-to-person transmission by direct contact (spread of droplets onto mucous membranes) or indirectly by infected blood, secretions, organs, semen and fomites. Risk is highest during the late stages of illness when the patient is vomiting, having diarrhoea or haemorrhaging. Risk during the incubation period is low. Under natural conditions, airborne transmission among humans has not been documented. Nosocomial infections have been frequent.
Incubation	Incubation period is usually 2–21 days.
Period of communicability	As long as blood, saliva, faeces and other secretions contain virus, which can be up to 6 months.

EPIDEMIOLOGY

Burden	Occurs in epidemics (see below).
Geographical distribution	Ebola outbreaks in Sudan have occurred in the southernmost area of the country, close to the border with Democratic Republic of the Congo.
Seasonality	No clearly evident seasonal pattern.
Alert threshold	One suspect case must lead to an alert.
Recent epidemics in the country	**2004 May–July/August**. Cases of acute haemorrhagic fever syndrome in Hai-Cuba, Yambio county, Western Equatoria, southern Sudan. A rapid assessment team is in the field to investigate the situation. The cases were confirmed to be of Ebola Haemorrhagic fever (EHF). A total of 17 cases and 7 deaths of EHF were reported, of which 13 were laboratory-confirmed and 4 epidemiologically linked. The last death was reported on 26 June in the Yambio hospital isolation ward. After a mandatory 42 days of viglant surveillance the outbreak was officially declared over on 7 August 2004. **1979 July–October**. On 2 August, a 45 year-old man was admitted to the N'zara hospital with a fever that had lasted for 3 days and recent onset of diarrhoea and vomiting. While at the N'zara hospital, he developed gastrointestinal haemorrhaging and died on 5 August. No precautionary isolation measures were taken or barrier-nursing techniques practised. Three of his relatives who had cared for him during his illness developed haemorrhagic fever and were hospitalized. All cases occurred among five families in a rural district in the remote savannah of southern Sudan. The district was later quarantined. The total number of cases was 34, of which 22 were fatal (CFR=65%). All cases were directly linked to the index case who was employed at the N'zara Cotton Manufacturing Factory. **1976 June–November**. The first case of EHF in Sudan was detected in N'zara (Western Equatoria, close to the border with Democratic Republic of the Congo) and then spread to Maridi, Tambura and Juba. On 27 June, a N'zara Cotton Manufacturing Factory cloth-room worker became ill with a haemorrhagic febrile disease and died in the N'zara hospital on 6 July. The disease was introduced to Maridi, 128 km away, by a case admitted to Maridi hospital. Spreading occurred mainly through close personal contact within the hospital. Several medical care personnel were infected, as transmission was usually associated with the act of nursing a patient. The viral subtype identified was named Ebola-Sudan (EBO-S). The total number of cases was 284 (the largest part in Maridi); the percentage of deaths among cases (case-fatality rate) was 53%.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	In case of outbreak, population movement can contribute to the spread of infection to non-affected areas. Contacts under daily follow-up should be encouraged to limit their movements through community sensitization and social mobilization.
Overcrowding	**Yes**	Prompt isolation of a suspect case is a key control strategy. All conditions favouring contact with sick persons, their cloths and bedding constitute a risk factor for increased transmission.
Poor access to health services	**Yes**	Health centres are essential as an alert network, not for providing treatment. Prompt identification of cases is paramount to rapidly implement control measures.
Food shortages	**No**	

Lack of safe water and poor sanitation	**No**	
Others	**No**	Activities related to hunting have been indicated as a risk factor for acquiring the infection on several occasions.
Risk assessment conclusions	The reservoir of Ebola haemorrhagic fever is not known, and it is therefore difficult to evaluate the risk of transmission. Implementation of control measures can also be difficult given cultural practices such as the custom of eating primate meat. With the exception of Uganda, Ebola outbreaks have always occurred in ecologically similar areas: these areas could represent the biotope of the reservoir. Moreover, there are indications that similar climatic patterns are associated with Ebola outbreaks. Monitoring climatic variables could therefore help to identify high-risk areas. Future priorities include identification of the reservoir in order to better target public health measures.	

PREVENTION AND CONTROL MEASURES

Case management	**Specific therapy:** not currently available for filoviral infections. **Supportive treatment:** – Analgesic drugs – Antimicrobial drugs (to avoid secondary infections) – Antimalarials (if clinically indicated) – Fluid replacement, with careful oral and less intravenous rehydration. **Implementation of barrier-nursing practices:** In order to prevent secondary infections, contact with the patient's lesions and body fluids should be minimized using standard isolation precautions: – Isolation of patients – Restriction of access to patients wards – Use of protective clothing – Safe disposal of waste – Disinfection of all non-disposable supplies and equipment – Safe burial practices. All the above measures can be implemented despite problems due to limited resources (see WHO/CDC*. Infection control for viral haemorrhagic fevers in the African care setting.* Geneva, WHO, 1998; WHO/EMC/EST/98.2).
Epidemic control	Epidemics of the disease in health care institutions with poor hygiene standards can be dramatically amplified through contact with patients or body fluids from infected patients (blood, vomitus, urine, stools, semen, saliva). The potential for explosive nosocomial infections constitutes the main threat to public health posed by the disease. Strict adherence to isolation precautions with all patients has been shown to reduce the risk of transmission. During the 1995 Ebola haemorrhagic fever outbreak in Kikwit (Democratic Republic of the Congo, 1995), no new cases were reported among health workers who used these precautions consistently.

Prevention	The following key elements are essential in the prevention of explosive epidemics in areas potentially subject to EHF: 1. Social mobilization and health education of the community, emphasizing: – Avoiding contact with body fluids of an EHF patient. – Seeking treatment early and avoiding harmful funeral practices. – Boiling and burning all clothing of an Ebola patient. – Use of protective methods when handling the patient and EHF patient's articles. – Avoiding consumption of dead animal meat found in the forest. 2. Correct case management, including barrier nursing and appropriate funeral practices. 3. Effective and efficient coordination of interventions. 4. Good logistics and security. 5. Vigilant surveillance, standard epidemiological practice and laboratory services. Health workers in EHF-prone regions should receive advance training in the use of isolation procedures and universal isolation precautions.

9. HIV/AIDS

DESCRIPTION

Infectious agent	Human immunodeficiency virus (HIV). Two types have been identified: HIV-1 and HIV-2; both have similar epidemiological characteristics. HIV-2 is less pathogenic than HIV-1.
Case definition	**AIDS case definition** Acquired immunodeficiency syndrome (AIDS) is the late clinical stage of HIV infection, defined as an illness characterized by one or more indicator diseases. **WHO staging system for HIV infection and disease in adults and adolescents** <u>*Stage 1*</u> 1. Asymptomatic. 2. Persistent generalized lymphadenopathy (PGL). Performance Scale 1: *asymptomatic, normal activity.* <u>*Stage 2*</u> 3. Weight loss, <10% of body weight. 4. Minor mucocutaneous manifestations (seborrheic dermatitis, prurigo, fungal nail infections, recurrent oral ulcerations, angular cheilitis). 5. Herpes zoster within the past 5 years. 6. Recurrent upper respiratory tract infections (e.g. bacterial sinusitis), And/or Performance Scale 2: *symptomatic, normal activity.* <u>*Stage 3*</u> 7. Weight loss, >10% of body weight. 8. Unexplained chronic diarrhoea, >1 month. 9. Unexplained prolonged fever (intermittent or constant), >1 month. 10. Oral candidiasis (thrush). 11. Oral hairy leukoplakia. 12. Pulmonary tuberculosis within the past year. 13. Severe bacterial infections (i.e. pneumonia, pyomyositis), And/or Performance Scale 3: *bedridden, <50% of the day during the past month.* <u>*Stage 4*</u> 14. HIV wasting syndrome, as defined by the US Centers for Disease Control and Prevention (CDC).[a] 15. *Pneumocystis carinii* pneumonia. 16. Toxoplasmosis of the brain. 17. Cryptosporidiosis with diarrhoea >1 month. 18. Cryptococcosis, extrapulmonary. 19. Cytomegalovirus (CMV) disease of an organ other than liver, spleen or lymph nodes. 20. Herpes simplex virus (HSV) infection, mucocutaneous >1 month, or visceral any duration. 21. Progressive multifocal leukoencephalopathy (PML). 22. Any disseminated endemic mycosis (e.g. histoplasmosis, coccidiomycosis). 23. Candidiasis of the oesophagus, trachea, bronchi or lungs. 24. Atypical mycobacteriosis, disseminated. 25. Non-typhoid *Salmonella* septicaemia. 26. Extrapulmonary tuberculosis. 27. Lymphoma. 28. Kaposi sarcoma. 29. HIV encephalopathy, as defined by CDC.[b] ***Note**: Both definitive and presumptive diagnoses are acceptable.* (a) HIV wasting syndrome: weight loss of >10% of body weight, plus either unexplained chronic diarrhoea (>1 month) or chronic weakness and unexplained prolonged fever (>1 month); (b) HIV **encephalopathy:** clinical finding of **disabling cognitive and/or motor dysfunction interfering with activities** of **daily living, progressing over weeks** to **months,** without a **concurrent illness or condition other than** HIV infection that could explain the findings.

	Expanded WHO case definition for AIDS surveillance* An adult or adolescent (aged >12 years) is considered to have AIDS if a test for HIV antibody gives a positive result, and one or more of the following conditions are present: 1. >10% body weight loss or cachexia, with diarrhoea or fever, or both, intermittent or constant, for at least 1 month, not known to be due to a condition unrelated to HIV. 2. Cryptococcal meningitis. 3. Pulmonary or extrapulmonary tuberculosis. 4. Kaposi sarcoma. 5. Neurological impairment that is sufficient to prevent independent daily activities, not known to be due to a condition unrelated to HIV infection (e.g. trauma or cerebrovascular accident). 6. Candidiasis of the oesophagus (which may be presumptively diagnosed based on the presence of oral candidiasis accompanied by dysphagia). 7. Clinically diagnosed life-threatening or recurrent episodes of pneumonia, with or without etiological confirmation. 8. Invasive cervical cancer. * WHO. *Weekly Epidemiological Record,* 1994, 69:273-275.
	Laboratory evidence of HIV This is most commonly based on detection of HIV antibody in serum samples using enzyme-linked immunosorbent assay (ELISA or EIA). When positive, this test must be confirmed with another test of higher specificity such as the Western blot, the indirect fluorescent antibody (IFA) test or a second ELISA test that is methodologically and/or antigenically independent. The rapid tests that are recommended by WHO have been evaluated at WHO collaborating centres and have levels of sensitivity and specificity comparable with WHO-recommended ELISA tests. The use of rapid HIV tests may afford several advantages in emergency and disaster settings, including: – Rapid tests that do not require refrigeration will be more suitable for remote and rural areas and sites without a guaranteed electricity supply. Long shelf-life is also important, especially for remote areas and sites performing smaller numbers of tests. – Many rapid tests require no laboratory equipment and can be performed in settings where electrical and water supplies need not be guaranteed. – Rapid tests can detect HIV antibodies in whole blood (finger-prick samples) as well as in serum/plasma, and testing may therefore be performed by non-laboratory personnel with adequate training and supervision.

Mode of transmission	Sexual intercourse (vaginal or anal) with an infected partner, especially in the presence of a concurrent ulcerative or non-ulcerative sexually transmitted infection (STI). Contaminated needles, syringes, other injecting equipment and injecting solutions (contamination often occurs when drug solutions are mixed or when multiple users draw up solutions from a single container). Transfusion of infected blood or blood products. Infected mother to her child during pregnancy, labour and delivery or through breastfeeding.
Incubation	Variable. On average, the time from HIV infection to clinical AIDS is 8–10 years, although AIDS may be manifested in less than 2 years or be delayed in onset beyond 10 years. Incubation times are shortened in resource-poor settings and in older patients. They can be prolonged by provision of primary prophylaxis for opportunistic infections or by antiretroviral treatment.
Period of communicability	Any person who is infected with HIV may pass the infection to another through the routes of transmission described above. Infectiousness is observed to be high during the initial period after infection. Studies suggest it increases further with increasing immune deficiency, clinical symptoms and presence of other STIs.

EPIDEMIOLOGY

Burden	**Estimated number of adults and children living with HIV/AIDS, end of 2001:** (including all people with HIV infection, whether or not they have developed symptoms of AIDS). Adults (15–49) 410 000 (2.6% of all adults) Women (15–49) 230 000 Children (0–15) 30 000 **Estimated number of deaths due to AIDS in 2001:** 23 000 **Reported AIDS cases in 2001:** 492 (Mode of transmission: heterosexual, 348; perinatal, 6; unknown, 138) **Estimated number of living orphans (2001):** 62 000 (Data source: WHO Sudan, 2004) In 2002, the results of an epidemiological survery conducted among 7, 385 individuals in 11 of the 16 sates, involving a number of groups at varying risk of infection were reported. Persons tested included Sudanese and nonSudanese. The seroprevalence among Sudanese was 1.6% . Of the 3, 355 women in ANC care, 30% (1.0%) were infected. Of 367 Sudanese sex workers, 16% (4.4%) were infected. Lesser prevalences were found among prisoners (4 of 200, 2.0%), soilders (2 of 377, 0.5%), those with STDs (4 of 362, 1.1%), university students (4 of 369, 1.1%) and TB patients (6 of 367, 1.6%). The Sudan National AIDS Control Programme HIV surveillance system indicates the large majority of finfections are acquired via heterosexual transmission, and this survey showed levels of awareness of HIV/AIDS and means to protect oneself from becoming infected were poor. **See:** UNAIDS - Sudan, Epidemiological fact sheet. 2004 update. http://www.unaids.org/html/pub/publications/fact-sheets01/sudan_en_pdf.pdf
Geographical distribution	No data available; HIV median prevalence among ANC attendees in 1998 was about 0.5% in urban areas and about 3.75% in rural areas.

Seasonality	Not applicable.
Alert threshold	One suspected case must be investigated.
Recent epidemics in the country	**Number of AIDS cases by year of reporting** 2003 : no data available 2002: no data available 2001 : 492 2000 : 652 1999 : 517 1998 : 511 1997: 270 1996 : 221 1995 : 257 1994: 201 1993: 191 1992: 184 1991: 188 1990: 130 1989: 122 1988: 64 1987: 2 1986: 2 1985: 0 1984: 0 1983: 0 1982: 0 1981: 0 1980: 0 1979: 0 **Total (end 2001):** 10959 (Data source: UNAIDS, 2004 update).

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	In emergency situations, population movement can: – Cause breakdown in family and social ties. – Erode traditional values and coping strategies. This can result in higher-risk sexual behavior, which increases the risk of HIV spread. – Influence illicit drug trafficking and drug use, which increases the risk of HIV transmission through injecting drug use.
Overcrowding	**Yes**	Groups with differing levels of HIV awareness, and differing rates of infection, are often placed together in temporary locations, such as refugee camps, where there is greater potential for sexual contact. Overcrowding can also influence injecting drug use patterns and result in increased risk of sharing contaminated injecting equipment (this has been noted in refugee camps).
Poor access to health services	**Yes**	Without adequate medical services, STIs, if left untreated in either partner, greatly increase the risk of acquiring HIV. Important materials for HIV prevention, particularly condoms, are likely to be lacking in an emergency situation. In emergency situations, services for drug dependence treatment usually do not exist. It is more likely to be difficult to access sterile injecting equipment.
Food shortages	**Yes**	The need for food is paramount in emergency situations, and exchanging sex for money to buy food and other essentials can occur (see "Sex work", below).
Lack of safe water and poor sanitation	**No**	

Others	**Yes**	**Sexual violence** Refugees and IDPs are often physically and socially powerless, with women and children at particular risk of sexual coercion, abuse or rape. Sexual violence carries a higher risk of infection because the person violated cannot protect herself or himself from unsafe sex, and because the virus can be transmitted more easily if bodily tissues are torn during violent sex. **Sex work** Exchange of sexual favours for basic needs such as money, shelter and security is common in or around refugee camps, and inevitably involves both the refugee and host communities. Both sex workers and clients are at risk of HIV infection if unprotected sex is practised. **Injecting drug use** In Sudan, no AIDS cases officially reported from the beginning of the epidemics to the end of 2001 had contracted the disease by injecting drugs. In typical emergency conditions, it is highly likely that drug injectors will be sharing needles, a practice that carries a very high risk of HIV transmission if one of the people sharing is infected. **Unsafe blood transfusions** Transfusion with HIV-infected blood is a highly efficient means of transmitting the virus. In emergency situations, when regular transfusion services have broken down, it is particularly difficult to ensure blood safety. **Adolescent health** Children in refugee settings may have little to occupy themselves with, which may lead them to experiment with sex earlier than children in other situations. **Lack of regular supplies** Lack of laboratory reagents for screening and testing, particularly for blood transfusions. Lack of condoms.

Risk assessment conclusions	HIV/AIDS is becoming an increasing problem in Sudan. Although the surveillance system is limited to four sites only, it is feared that Sudan could be facing a generalized epidemic. HIV prevalence among pregnant women was reported to be 2.94% in 1997. Based on recent HIV surveillance data in Khartoum State in 2000, 2.5% of women attending gynaecological clinics were found to be HIV seropositive, compared with HIV seropositivity rates of 1.86% in pregnant women attending antenatal clinics. HIV rates among blood donors increased from 0.15% in 1993 to 1.4% in 1999–2000. There is a clear link between HIV and tuberculosis (TB). In 1999, HIV prevalence among TB patients varied between 7.7% and 20% depending on the regions. Women attending both gynaecological and antenatal clinics in Khartoum State showed STI incidence of 10.5% and 34% respectively. **Reported AIDS cases by mode of transmission (from the beginning of the epidemic* up to end 2001):** Heterosexual contacts 3758 (93.9%) Blood and blood products 12 (0.3%) Perinatal 96 (2.4%) Unknown 138 (3.4%) All AIDS cases reported 4004 (100%) * The earliest AIDS cases in Sudan were reported in 1986 (Source: UN AIDS Sudan epidemiological profile fact sheet on HIV/AIDS and sexually transmitted diseases, 1 September 2004).
	All stakeholders involved in humanitarian activities must be sensitized to the importance of addressing HIV in tandem with all other activities. Activities should include HIV prevention (promotion of safer sexual behaviours, treatment of STIs, blood safety) and care and support for people living with HIV/AIDS (PLWHA). They must reach vulnerable populations and address the needs of women and children. All stakeholders must also be sensitized about HIV risks associated with injecting drug users and the need for drug dependence treatment and risk reduction education and counselling.

PREVENTION AND CONTROL MEASURES

Case management	Provide high-quality care and support to all PLWHA, which includes counselling, psychosocial support, treatment for opportunistic infections (e.g. TB), palliative care and access to antiretroviral therapy where feasible. Support PLWHA to live normal and productive lives that are free of stigmatization and discrimination.
Prevention	**Reduce sexual and mother-to-child transmission** *Awareness and life skills education*, especially among youth, to ensure that all people are well informed of what does, and does not, constitute a mode of transmission; of how and where to acquire free condoms and medical attention if necessary; and information on basic personal hygiene. *Condom promotion* to ensure that good-quality condoms are freely available to those who need them, using culturally sensitive instructions and distribution mechanisms. *STI control,* including for sex workers, using the syndromic STI management approach, with partner notification and promotion of safer sex. *Reduce mother-to-child transmission of HIV* by: – the primary prevention of HIV among women, especially young women – avoiding unintended pregnancies among HIV-infected women and promoting family planning methods, particularly in women who are infected with HIV – preventing HIV transmission from infected pregnant women to their infants by: – using an antiretroviral prophylaxis regimen; – avoiding unnecessary and invasive obstetrical procedures such as artificial rupture of membranes or episiotomy; and – modifying infant feeding practices (replacement feeding given with a cup when acceptable, feasible, affordable, sustainable and safe; otherwise exclusive breastfeeding for the first six months of life is recommended: See*The optimal duration of exclusive breastfeeding - A systematic review,* WHO/FCH/CAH/01.23). **Blood safety** HIV testing of all transfused blood. Avoid non-essential blood transfusion – Recruitment of safe blood donor pool. **Prevention among injecting drug users** Ready access to sterile needles, syringes and other injecting equipment (and disposal of used equipment). HIV risk reduction education and counselling for injecting drug users (including peer outreach when possible). Drug dependence treatment services, including substitution treatment (e.g. methadone) where possible. Access to STI and HIV/AIDS treatment for injecting drug users.

	Universal precautions Washing hands thoroughly with soap and water, especially after contact with body fluids or wounds. Using protective gloves and clothing when there is risk of contact with blood or other potentially infected body fluids. Safe handling and disposal of waste material, needles and other sharp instruments. Proper cleaning and disinfection of medical instruments between patients. **Physical protection** The protection of the most vulnerable, especially women and children, from violence and abuse is not only an important principle of human rights but is also essential for reducing the risk of HIV infection.
Protecting health care workers	In order to reduce nosocomial transmission, health workers must strictly adhere to universal precautions with all patients and laboratory samples – whether or not known to be infected with HIV. Health care workers should have access to voluntary counselling, testing and care; those deployed in complex emergencies frequently experience significant occupational stress, and those tested as part of the management of occupational exposures will require additional support.
Counselling and voluntary testing programmes	The establishment of voluntary counselling and testing services to help individuals make informed decisions about HIV testing should be considered when relative stability has been restored. Displaced populations are often coerced into testing or are required to make decisions about testing when they are suffering acute or post-traumatic stress disorders. As displaced populations are often tested before resettlement in other countries, it is critical that they receive counselling on the legal and social implications of the test. Often, migration or temporary residency status is contingent on the applicant's having HIV antibody seronegative status. Post-test counselling is essential for both seronegative and seropositive results. Displaced populations and conflict survivors who are already traumatized will require additional psychosocial support if they test seropositive. Typically, the support networks of displaced persons are disrupted, and suicide risk assessment forms an important part of post-test counselling in a refugee or conflict context. Testing of orphaned minors should be done with the consent of their official guardians only where there is an immediate health concern or benefit to the child. **No mandatory screening should take place before admittance to substitute care**.
Immunization	Asymptomatic HIV-infected children should be immunized with the EPI vaccines. Symptomatic HIV-infected children should NOT receive BCG or yellow fever vaccine.

10. LEISHMANIASIS (CUTANEOUS AND MUCOSAL)

DESCRIPTION

Infectious agent	Protozoan, belonging to the genus *Leishmania:* • *L. major*, agent of cutaneous leishmaniasis (and, less frequently, of mucosal leishmaniasis) • *L. donovani*, agent of mucosal leishmaniasis (see *Visceral leishmaniasis*).
Case definition	**Clinical description** Cutaneous leishmaniasis is characterized by the appearance of one or more skin lesions, typically on uncovered parts of the body; the face, neck, arms and legs are the most common sites. A nodule may appear at the site of inoculation and may enlarge to become an indolent ulcer. The sore may remain at this stage for a variable time before healing, typically leaving a depressed scar. Other atypical forms may occur. In some individuals, certain strains can disseminate and cause mucosal lesions. These sequelae involve nasopharyngeal tissues and can be very disfiguring with major psychological consequences (see below). Sudanese mucosal leishmaniasis is a chronic infection of the upper respiratory tract and/or oral mucosa caused mainly by *L.donovani* or, less frequently, by *L.major*. The disease occurs in areas of the country endemic for visceral leishmaniasis. The condition may develop during or after an attack of visceral leishmaniasis, but in most cases it is a primary mucosal disease. It is not preceded or accompanied by a cutaneous lesion. The duration of the disease can vary between a few months and several years. **Laboratory criteria** • Positive parasitology (stained smear or culture from the lesion) Positive serology (immunofluorescent assay, ELISA, Direct Agglutination Test) for mucosal leishmaniasis only. **WHO operational definitions** • A case of cutaneous leishmaniasis can be defined as a person showing clinical signs (skin lesions) with parasitological confirmation of the diagnosis (positive smear or culture). • A case of mucosal leishmaniasis can be defined as a person showing clinical signs (mucosal lesions) with parasitological confirmation of the diagnosis and/or
Mode of transmission	From the reservoir host through the bite of infective female phlebotomines (sandflies). *Phlebotomus papatasi* is the vector of *L. major* in Sudan. The highest vector population density is usually found when the temperature is high, humidity is medium and rainfall is low. The vector is domestic and peridomestic in the villages. Humans are the preferred hosts, and daily biting activity is highest in the evening. There is limited information on the animal reservoir of cutaneous leishmaniasis in Sudan. It is probably represented by the Nile rat *Arvicanthis niloticus*. *Phlebotomus orientalis*, the vector of *L. donovani* in Sudan, is most abundant where *Acacia seyal* and *Balanites aegyptica* vegetation is common and where the soil is rich in clay.

Incubation	**Cutaneous leishmaniasis:** usually 2–4 weeks but may be longer. The incubation period is inversely proportional to the size of the inoculum. **Mucosal leishmaniasis:** difficult to establish. Patients usually provide no history of cutaneous leishmaniasis. Some patients give a history of treated visceral leishmaniasis; others may have had mucosal and visceral leishmaniasis concurrently.
Period of communicability	An infected subject is susceptible to transmit the parasite as long as it remains in lesions; in untreated cases, usually a few months to 2 years.

EPIDEMIOLOGY

Burden	**Year** **Number of cases** **Deaths** 2000 204 2 2001 351 32 2002 258 --- (Data source: WHO/Sudan, 2004)
Geographical distribution	Darfur and Kordofan provinces are known to be endemic for zoonotic **cutaneous leishmaniasis.** Epidemics in recent years have occurred in Northern, Eastern, Khartoum and Central provinces. Most patients with **mucosal leishmaniasis** come from areas also endemic for visceral leishmaniasis.
Seasonality	During recent epidemics in Sudan, the peak of infection was believed to occur in August and December, dropping sharply in March, April and May.
Recent epidemics in the country	**1976** Shendi–Atbara area (Northern Province). **1985–1987** Khartoum Province: about 10 000 recorded cases. Peak incidence in September 1986. **1990–1992** Dongola and Mahas areas (Northern Province).

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Movements of population such as displaced populations contribute to the maintenance of epidemics, bringing non-immune people into endemic areas and infected people into non-endemic areas where the vector is widespread. The characteristics of epidemics in Sudan are typical of a disease newly introduced into a previously non-immune population, in that all age groups have been affected. Migrants from western Sudan (endemic for zoonotic cutaneous leishmaniasis) to Khartoum Province probably contributed to the epidemic that affected this area between 1985 and 1987.
Overcrowding	**Yes**	Overcrowding can increase the risk of contact with animal reservoir.
Poor access to health services	**Yes**	Lack of treatment increases the number of infected individuals in the population.
Food shortages	**Yes**	However, malnourished people are more susceptible to the infection due to a weakened immune response. Many of the patients seeking treatment are also malnourished.

Lack of safe water and poor sanitation	**Yes**	Open sewage systems and lack of garbage or rubble collection favour the proliferation of vector breeding sites.
Others	**Yes**	Rapid expansion of towns and villages, establishment of new settlements and consequent development of previously uninhabited areas. Interruption of control measures: discontinuation of insecticide spraying for malaria control in Sudan in the years preceding the epidemics may have contributed to an increase in the vector population. Increase in the rodent population: coinciding, in 1976 and 1986, with the two epidemics of cutaneous leishmaniasis in Sudan. Prolonged low rainfall: the dry soil cracks and becomes waterlogged, creating ideal breeding conditions for the sandfly.
Risk assessment conclusions		The disease is known as *hashara* ("insect") in Sudan.

PREVENTION AND CONTROL MEASURES

Case management	**Cutaneous leishmaniasis** is a self-limiting disease. Self-healing usually occurs within 6 months, but skin scarring and changes in pigmentation always follow. There is currently no uniform protocol for treating cutaneous leishmaniasis in Sudan. Patients with minor lesions are usually reassured and left to heal spontaneously. Patients with severe or multiple lesions (>5), diabetics with lesions and patients who acquired infection in geographic regions where mucosal disease has been reported should be treated promptly. Treatment is based on: Pentavalent antimonials (the drug of choice in Sudan is sodium stibogluconate) as a first-line drug, except when resistance develops. The drugs can be administered systemically (IM or IV), or locally (intralesional infiltrations). WHO recommends the following course: 20 mg/kg per day for 20 days. In the presence of resistance, second-line drugs must be used. Standard amphotericin B, aminosidine plus pentavalent antimonials or pentamidine isetionate are the main alternatives. Other therapeutic options are available: – Antifungal drugs (e.g. ketoconazole). – Cryotherapy. Patients with **mucosal leishmaniasis** respond well to treatment with pentavalent antimony compounds (sodium stibogluconate).
Epidemic control	Epidemics of cutaneous leishmaniasis can be controlled by an integrated, feasible and efficient strategy based on: — **Provision of first-line drug** (pentavalent antimonials) to improve cure rate. — **Provision of long-lasting bednets** (insecticide-treated nets – ITNs) to limit contact between human and vector. — **Health education and social interventions** to increase awareness and improve early diagnosis.

Prevention	**Personal protective measures** are effective in preventing contact between sandflies and humans. Such measures include skin repellents, vaporizing liquids, bednets impregnated or sprayed with pyrethroids, and screened doors and windows. **Vector control:** application of residual insecticides on surfaces where sandflies rest, such as indoor and outdoor walls, tree trunks, rock crevices, water wells and flowering plants may be effective in reducing the size of the sandfly population over time, thus decreasing the risk of infection. However, this measure is not recommended since it produces a transient effect only. **Reservoir control:** control methods must be adapted to the biology of the reservoir species (anticoagulants, poison baits, deep ploughing to eliminate plants on which the rodents feed, use of artificial canals or barriers to prevent colonization or reinvasion). No definitive control method against *Arvicanthis* is currently known. Many field activities for control of cutaneous leishmaniasis are integrated with those for malaria control. Work is in progress to evaluate the use of mosquito nets impregnated with insecticide to reduce human–fly contact.

11. VISCERAL LEISHMANIASIS (KALA AZAR)

DESCRIPTION

Infectious agent	Protozoan: *Leishmania donovani*
Case definition	**Clinical description** An illness with prolonged irregular fever, splenomegaly and weight loss as its main symptoms. • Post–kala-azar dermal leishmaniasis (PKDL) is increasingly recognized in Sudan as a complication of visceral leishmaniasis, occurring in about 55% of patients during treatment or within 0–6 months after treatment. It is characterized by a rash that may be macular, maculopapular, nodular or plaque-like. • Sudanese mucosal leishmaniasis is a chronic infection of the upper respiratory tract and/or oral mucosa caused mainly by *L. donovani* (see *Cutaneous leishmaniasis).* The disease occurs in areas of the country endemic for visceral leishmaniasis. In most cases it is a primary mucosal disease, not preceded or accompanied by a cutaneous lesion, but less frequently the condition may develop during or after an attack of visceral leishmaniasis. In this case the disease represents a phenomenon similar to PKDL. **Laboratory criteria** • Positive parasitology. – stained smears from bone marrow, spleen, liver, lymph node, blood or, – culture of the organism from a biopsy or aspirated material. • Positive serology (immunofluorescent assay, ELISA, Direct Agglutination Test). **WHO operational definition** • A case of visceral leishmaniasis (VL) is a person showing clinical signs (prolonged irregular fever, splenomegaly and weight loss) with serological (at peripheral geographical level) and/or (when feasible at central level) parasitological confirmation of the diagnosis. The main differential diagnosis is malaria. In endemic malarious areas, VL must be suspected when fever lasts for more than 2 weeks and no response has been achieved with antimalarial drugs (assuming drug-resistant malaria has also been considered).
Mode of transmission	Vector-borne, through the bite of infective female phlebotomines (sandflies). *Phlebotomus orientalis*, the vector of *L. donovani* in Sudan, is most abundant where *Acacia seyal* and *Balanites aegyptica* vegetation is common and where the soil is rich in clay. Transmission dynamics have not been elucidated fully; the large numbers of patients with PKDL in heavily affected villages indicate a human reservoir and anthroponotic transmission, whereas heavy transmission in scarcely populated areas suggests zoonotic transmission.
Incubation	Usually 2–6 months. Intensity of infection, partial immunity resulting from previous exposure, intercurrent illness, malnutrition and other factors may play a role in determining the acuteness or slowness of the course.
Period of communicability	An infected subject is susceptible to transmit the parasite to sandflies as long as it persists in the circulating blood or skin. Infectivity for sandflies may persist even after clinical recovery of human patients.

EPIDEMIOLOGY

Burden	**Number of cases reported (July–June):** **2001–2002:** 2413 **2000–2001:** 2308 **1999–2000:** 3922 **1998–1999:** 4804 **1997–1998:** 6182 (Data Source: WHO/Sudan, 2004)
Geographical distribution	The disease is historically endemic in a wide belt extending from the western bank of the White Nile and the Sudan–Ethiopian border. This area includes the southern areas of Central and Eastern provinces and the north-eastern part of Upper Nile Province. Since 1989, visceral leishmaniasis has spread in the western part of Upper Nile Province, where visceral leishmaniasis has never been reported before. Cases have also been reported from Darfur Province in west Sudan, the Nuba mountains area (Kordofan Province) and the Kapoeta area in Eastern Equatoria.
Seasonality	It is likely that the peak of transmission occurs towards the end of the dry season (March–June), especially just before the rainy season begins (May, June), when large numbers of *P. Orientalis* appear. Most individuals report with illness after the rains in October and November. **Number of cases reported in the period 1997–2002 (monthly basis):** **January:** 2335 **July:** 934 **February:** 2094 **August:** 870 **March:** 1883 **September:** 1083 **April:** 1554 **October:** 1856 **May:** 1376 **November:** 2384 **June:** 1122 **December:** 2138 (Data source: WHO/Sudan)
Recent epidemics in the country	**2002**. A severe increase in the number of VL cases was reported in October and November from communities in southern Sudan. The overlap of areas affected by VL and areas of conflict suggests that insecurity, malnutrition and poor access to health care lower people's natural resistance, creating an epidemic-prone environment. **1997–1998**. A dramatic upsurge in the number of VL cases was reported from eastern Sudan (Atbara river area): more than 2500 confirmed cases were registered in a MSF-run treatment centre in Gedaref State from October to December 1997 (+439% compared with the same period in 1996). Cumulative factors played a role: influx of displaced and non-immune population from southern states and overall decline in the nutritional status of the population. **1984–1994**.Upper Nile Province (southern Sudan): because of the isolation of the area caused by the war, the epidemics went unnoticed until 1988. Between 1984 and 1994, an estimated 100 000 died. A number of causes were proposed for this outbreak in a previously non-endemic area: • Regeneration of *Acacia seyal* and *Balanites aegyptica* forests after they were destroyed in the 1960s. • Introduction of the parasite from VL-endemic areas along the Ethiopian border by movement of military personnel. • Discontinuation of residual insecticide spraying for malaria because of the war. • Malnutrition of the people. From 1990 onwards, the epidemics reached the southern part of Kordofan Province and, in 1995–1996 the eastern part of Upper Nile Province.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Movements of displaced populations contribute to the maintenance of epidemics, bringing non immune people into endemic areas and infected people into non-endemic areas where the vector is widespread. The characteristics of epidemics in Sudan are typical of a disease newly introduced into a previously non-immune population: all age groups are affected. Migrants from western Sudan (endemic for zoonotic cutaneous leishmaniasis) to Khartoum Province probably contributed to the epidemics.
Overcrowding	**Yes**	Persons with PKDL act as reservoirs of *Leishmania* parasites in anthroponotic foci.
Poor access to health services	**Yes**	As a result of many factors (geographical, economic and cultural, poor transportation). Additionally, since most services are not free of charge, most patients seek traditional treatment measures.
Food shortages	**Yes**	Poor nutritional status increases susceptibility to VL infection and disease.
Lack of safe water and poor sanitation	**No**	
Others	**Yes**	*Acacia seyal* and *Balanites aegyptica* woodland: the disease vector is found only in forests where these trees are present and in villages in or near these forests. Black cotton soils: this clay-rich soil shrinks and swells with drying and wetting to the extent that very few woody plants can survive in it. However, certain trees survive and even thrive in it, including *A. seyal* and *B. aegyptica*. Termitaria are thought to provide the insects with a relatively low, stable temperature and high humidity during the heat of the day. Termitaria have been found to be resting sites for *P. martini* in south-eastern Sudan, but many localities harbouring *P. orientalis* have no or small termitaria. Interruption of control measures: discontinuation of insecticide spraying for malaria control in Sudan may have led to an increase in the vector population and contributed to the VL epidemics in southern Sudan. Prolonged low rainfall: the dry soil cracks and becomes waterlogged, creating ideal breeding/resting sites for the sandfly. *P. orientalis* is most abundant towards the end of the dry season (March–June).
Risk assessment conclusions		Visceral leishmaniasis is endemic in Sudan: the first case was reported in 1904. High mortality in the country is mainly due to the absence of diagnostic facilities, the unavailability of first-line drugs at the peripheral level and increasing resistance to pentavalent antimonials. The majority of cases are found in children and teenagers (up to 75%) as transmission occurs early in life. Prognosis is usually severe due to high prevalence of malnutrition and associated diseases - such as TB and respiratory and/or intestinal infections.

PREVENTION AND CONTROL MEASURES

Case management	Visceral leishmaniasis is a severe and fatal disease without treatment. **First-line treatment:** pentavalent antimonials (Sodium stibogluconate was introduced in Sudan in 1947). WHO recommends the following course: 20 mg/kg per day for 30 days. **Second-line treatment:** liposomal amphotericin B, aminosidine. Three lipid-associated amphotericin B formulations (liposomal, colloidal dispersion, lipid complex) are highly effective against visceral leishmaniasis and better tolerated than the conventional preparation. Liposomal amphotericin B is an expensive drug and cannot therefore be recommended as a first-line drug; if available, it should be the drug of choice in cases resistant or unresponsive to antimonials due to its high effectiveness and lower toxicity. Treatment for PKDL is needed only for those who have severe and prolonged disease; sodium stibogluconate is usually sufficient. Liposomal amphotericin B is also effective. Patients with mucosal leishmaniasis respond well to treatment with pentavalent antimony compounds (sodium stibogluconate). Resistance to pentavalent antimonials has been reported from eastern Sudan, but data are fragmentary. It has not been reported from southern Sudan.
Epidemic control	Epidemics of visceral leishmaiasis can be controlled by an integrated, feasible and efficient strategy based on: — Provision of first-line drug (pentavalent antimonials) to improve cure rate and, in zoonotic foci, reduce transmission. — Provision of insecticide-treated nets (ITNs) to limit contact between human and vector. Long-lasting ITNs are now available. — Health education and social interventions to increase awareness and improve early diagnosis, early health-seeking and good treatment compliance. Visceral leishmaniasis control programmes may be integrated with malaria control programmes.
Prevention	**Personal protective measures** are effective in preventing contact of sandflies and humans. Such measures include skin repellents, vaporizing liquids, bednets impregnated or sprayed with pyrethroids, and screened doors and windows. Usually, the mesh used for leishmaniasis control is the same as that used for malaria control: it therefore has to be impregnated with insecticide, otherwise sandflies will pass through. **Vector control:** application of residual insecticides on surfaces where sandflies rest, such as indoor and outdoor walls and tree trunks, could be effective in reducing the size of the sandfly population over time and thus decrease the risk of infection, but is not recommended due to its high cost, low sustainability and logistic constraints. **Reservoir control:** The animal reservoir for *L. donovani* has not yet been identified. **Systematic case detection and rapid treatment:** this applies to anthroponotic foci. Many programmes and field activities are integrated with those for malaria control. Work is in progress to evaluate the use of vaccine against *L. donovani* and of mosquito nets impregnated with insecticide to reduce human–fly contact.

12. LEPROSY

DESCRIPTION

Infectious agent	Bacterium: *Mycobacterium leprae*.
Case definition	**WHO operational definition:** A case of leprosy is defined as a person showing hypopigmented or reddish skin lesion(s) with definite loss of sensation. The operational case-definition includes: • Retrieved defaulters with signs of active disease. • Relapsed cases who have previously completed a full course of treatment. **Case classification (clinical):** Paucibacillary leprosy: 1–5 patches or lesions on the skin. Multibacillary leprosy: more than 5 patches or lesions on the skin. **Laboratory criteria for confirmation:** In practice, laboratories are not essential for the diagnosis of leprosy.
Mode of transmission	Not clearly established: organisms probably enter the human body through the mucous membranes of the upper respiratory tract and possibly through broken skin during close and frequent contact with untreated, infected persons.
Incubation	9 months to 40 years; on average 3–4 years.
Period of communicability	• If not treated: infectivity is possible, the risk being higher for contacts of multibacillary cases than for paucibacillary cases. • Treated: infectivity vanishes within a few doses of treatment with multidrug therapy (MDT).

EPIDEMIOLOGY

Burden

Registered cases at the end of 2003 (point prevalence): 999

New cases detected (2003) = 906 (791 MB; 175 PB).
Overall prevalence (2003) = 0.35/10 000 population.

Year	New cases	Prevalence/10 000 population
1992	484	0.3
1995	3800	2.0
1996	4620	2.6
1997	3633	1.3
1998	2077	0.9
1999	2426	0.7
2001	1299	0.4
2002	1361	0.4
2003	906	0.35

Geographical distribution

Cases of leprosy are widespread throughout the country.

New cases (2001)

Khartoum	175 (151 MB; 24 PB)
Northern	20 (13 MB; 7 PB)
Eastern	38 (18 MB; 20 PB)
Kordofan	110 (90 MB; 20 PB)
Darfur	263 (168 MB; 95 PB)
Central	395 (220 MB; 175 PB)
Bahr Al Ghazal	234 (180 MB; 54 PB)
Equatoria	36 (32 MB; 4 PB)
Upper Nile	28 (20 MB; 8 PB)

The highest prevalance occurs in Darfur Province, with approximately 0.9 per 10 000 population in 2002, followed by the central zone, Bahr Al Ghazal, Equatoria/Kordofan zones and Khartoum. The northern and southern zones have the lowest prevalence. However, lower prevalence in the southern states reflects weaker leprosy programme activities due to insecurity, while that in the northern zones is realistic.

Seasonality

No seasonality registered.

Recent epidemics in the country

The disease has no epidemic potential.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**No**	
Overcrowding	**Yes**	Close contact facilitates transmission. However, reducing physical contact is of dubious value and can lead to stigmatization.
Poor access to health services	**Yes**	Lack of treatment increases the number of infected individuals in the population.
Food shortages	**No**	

Lack of safe water and poor sanitation	**No**	
Others	**No**	
Risk assessment conclusions		At the end of 2003, with a prevalence rate of 0.5/10.000, Sudan was not among the countries in which leprosy is considered a public health problem (prevalence rate >1 per 10 000 and population more than 1 million). However, Sudan is the country with the second highest leprosy burden in EMRO after Egypt. Data are likely to be incomplete due to lack of coverage in southern Sudan. In the effort to eliminate leprosy, a particular approach is needed for southern states of Sudan because of the ongoing complex emergency situation, with implementation of MDT in as many areas as possible, sound involvement of NGOs, community sensibilization and direct involvement in case-finding and case management. Differential diagnosis between leprosy and post–kala-azar dermal leishmaniasis should always be considered, since any form of leprosy may be confused with PKDL. However, if the three cardinal signs of leprosy are always kept in mind (anaesthetic lesions, nerve enlargement and the demonstration of *M. leprae),* no confusion between PKDL and leprosy should arise.

PREVENTION AND CONTROL MEASURES

Case management	Treatment by multidrug therapy (MDT) according to case classification: **Multibacillary leprosy**: the standard regime is a combination of the following for 12 months: **Adults:** – **Rifampicin:** 600 mg once a month. – **Dapsone:** 100 mg once a day. – **Clofazimine:** 50 mg once a day and 300 mg once a month. **Children** must receive appropriately scaled-down doses (in child blister-packs). **Paucibacillary leprosy**: the standard regimen is a combination of the following for 6 months: **Adults:** – **Rifampicin:** 600 mg once a month. – **Dapsone:** 100 mg once a day. **Children** must receive appropriately scaled-down doses (in child blister-packs). A core element of the elimination strategy is to make leprosy diagnosis and MDT available at all health centres, to **all** existing leprosy patients. MDT is provided free of charge by WHO.
Prevention	Early detection and treatment of cases.
Immunization	BCG vaccination can induce limited protection against the tuberculoid form of the disease in some populations. However, this is one of the control methods against tuberculosis and must not be undertaken specifically against leprosy.

13. LYMPHATIC FILARIASIS

DESCRIPTION

Infectious agent	Helminth: *Wuchereria bancrofti*, a filarial worm belonging to the class *Nematoda*.
Case definition	**Clinical case definition:** Hydrocele or lymphoedema in a resident of an endemic area for which other causes of these findings have been excluded. **Laboratory criteria for diagnosis:** Positive parasite identification by: – Direct blood examination **or** – Ultrasound **or** – Positive antigen-detection test. **Case classification:** – **Suspected:** Not applicable. – **Probable:** A case that meets the clinical case definition. – **Confirmed:** A person with positive laboratory criteria even if he or she does not meet the clinical case definition. The burden of lymphatic filariasis, as measured in disability-adjusted life years (DALYs), is the highest of all tropical diseases after malaria.
Mode of transmission	Bite of infected blood-feeding female mosquitoes (mainly *Anopheles* spp.; also *Culex* spp.), which transmit immature larval forms of the parasitic worms from human to human.
Incubation	1 month to 1 year and more: recidivant attacks of "filarial fever" (pain and inflammation of lymph nodes and ducts, often accompanied by fever, nausea and vomiting). 5 to 20 years: chronic illness manifestations may include elephantiasis (massive swelling of limbs), hydrocele (swelling of the scrotum in males), enlarged breasts in females and chyluria.
Period of communicability	As long as *microfilariae* are present in the peripheral blood (from 6–12 months to 5–10 years after the infective bite).

EPIDEMIOLOGY

Burden	Sudan is included in the Afrotropical endemic region. The estimated population at risk is 12.2 million.
Geographical distribution	Lymphatic filariasis is endemic in the southern part of the country. It is not present in the northern part. Lymphatic filariasis is a focal disease, and an important feature is that it tends to occur in circumscribed zones. Observed prevalence rates vary greatly from one geographical area to another, and even between villages within the same district.
Seasonality	Seasonal pattern is not clearly evident.
Recent epidemics in the country	The disease is not epidemic-prone.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Disease-free population can be displaced in endemic areas.
Overcrowding	**Yes**	Population crowding increases the risk of transmission.
Poor access to health services	**Yes**	Increased risk of transmission, especially where no services exist for providing treatment for lymphatic filariasis.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	Providing safe water is a secondary preventive measure (prevention of the disease, not of the infection) since it enables some of the hygienic measures recommended for the affected body parts. Poor sanitation may contribute to create breeding sites for mosquito vectors (especially *Culex* spp).
Others	**Yes**	Established link between the grade of poverty and the prevalence of LF.
Risk assessment conclusions	The complex emergency situation in Sudan is one of the reasons why this country's inclusion in the Global Programme to Eliminate Lymphatic Filariasis (GPELF) has been delayed. This poses a risk for elimination of lymphatic filariasis in neighbouring countries since Sudan can represent a source of transmission. Health Mapping for lymphatic filariasis in order to localize precisely populations at risk in Sudan has begun and should be completed soon. It will then be possible to implement the control programme, monitor drug coverage over time and eliminate the disease in space and time. GPELF in Sudan will bring “beyond filariasis" benefits: for example, albendazole is also an effective and safe drug for treating soil-transmitted helminth infections; ivermectin is also effective against many intestinal parasites, scabies and lice.	

PREVENTION AND CONTROL MEASURES

Case management	• **Hygiene measures** for the affected body parts (and, when necessary, antibiotics and antifungal agents) can decrease the risk of adenolymphangitis: – Washing the affected parts twice daily with soap and water. – Raising the affected limb at night. – Exercising to promote lymph flow. – Keeping nails short and clean. – Wearing comfortable footwear. – Using antiseptic or antibiotic creams to treat small wounds or abrasions or, in severe cases, systemic antibiotics. • **Drug regimen** – Diethylcarbamazine (DEC) 6 mg/kg single dose for 12 days. However, a single 6 mg/kg dose is equally effective in killing the adult worm and in reducing microfilaraemia. – DEC 6–8 mg/kg per day for 2 days each month for 12 months. Since the use of DEC in patients with either onchocerciasis or loiasis can be unsafe, it is important that patients with bancroftian filariasis who live in areas endemic for these other infections be examined for coinfection with these parasites before being treated with DEC. Alternatively, ivermectin and albendazole can be used: ivermectin, though very effective in decreasing microfilaraemia, appears not to kill adult worms (i.e. it is not macrofilaricidal) and thus does not cure infection completely. Albendazole can be macrofilaricidal for *W. bancrofti*, but optimization of its usage has not been attempted.
Prevention and Control	Prevention of infection can be achieved only by reducing contact between humans and vectors or by decreasing the amount of infection the vector can acquire. **A – Population level:** Even when effective mosquito control can be implemented, the long lifespan of the parasite (4–8 years) means that the infection remains in the community for a long period of time, generally longer than the period over which intensive vector control efforts can be sustained. The recent advent of the extremely effective single-dose, once-yearly drug regimen has permitted an alternative approach – and the launch of GPELF in 1998. When a country is included in the GPELF, the following steps are undertaken: 1. The national territory is divided into areas called implementation units (IUs). 2. In IUs known to be endemic, mass drug administration (MDA) is implemented if the prevalence by antigenaemia in the IU exceeds 1%. 3. In each IU where lymphatic filariasis status is uncertain, a village will be selected that has the greatest likelihood of transmission (or will be randomly selected if no information available). – In the selected villages, a sample of 250 persons aged 15 and older should be examined using the ICT card test. If any person has a positive result, the IU should be classified as endemic. – For each village, the number of persons examined and the number of persons positive is required for calculation of the prevalence. – MDA will be implemented if the prevalence in the IU exceeds 1%.

	GPELF has two main goals: to interrupt transmission of infection and to alleviate and prevent suffering and disability caused by the disease: **1. To interrupt transmission of infection**, the entire at-risk population must be treated for a period long enough to ensure that levels of microfilariae in the blood remain below those necessary to sustain transmission. Therefore, *a yearly, 1- dose* regimen (mass drug administration or MDA) of the following drugs must be given: Areas with concurrent onchocerciasis: Albendazole 400 mg + ivermectin 150 µg/kg of body weight once a year for 4–6 years. Areas with no concurrent onchocerciasis: Albendazole 400 mg + DEC 6 mg/kg of body weight once a year for 4–6 years, or DEC-fortified salt for daily use for at least 12 months. In areas with *concurrent loiasis*, systematic mass interventions cannot currently be envisaged because of the risk of severe adverse reactions in patients with high-density *Loa loa* infections (about 1 in 10 000 treatments). **2. To alleviate and prevent suffering and to reduce the disability and handicap caused by the chronic consequences of lymphatic filariasis,** the principal strategy focuses on: (1) increasing lymph flow through elevation and exercise of the swollen limb; (2) decreasing secondary bacterial and fungal infections of limbs or genitals where lymphatic function has already been compromised by filarial infection. Secondary infection is the primary determinant of the worsening of lymphoedema and elephantiasis. Scrupulous hygiene and local care are dramatically effective in preventing painful, debilitating and damaging episodes of lymphangitis. These consist of regular washing with soap and water, daily exercising of the limbs, wearing of comfortable footwear and carrying out other simple procedures at home, and at a very low cost (see *Case management* for details). Whereas MDA can be generally expected to reduce or interrupt transmission of LF, the goal of GPELF could be achieved more rapidly through additional vector control in some situations. Where MDA coverage rates or duration are limited, the added impact of effective vector control can most usefully augment the GPELF. **B – Individual level:** Lymphatic filariasis vectors usually bite between the hours of dusk and dawn. Contacts with infected mosquitoes can be reduced through the use of repellents, bednets or insecticide-impregnated materials.
Epidemic control	Relatively low infectivity and long incubation make outbreaks of lymphatic filariasis unlikely.

14. MALARIA

DESCRIPTION

Infectious agent	In Sudan, about 90% of all malaria cases are caused by the protozoan parasite *Plasmodium falciparum.* This causes the most life-threatening form of the disease. *P. vivax* and *P. ovale* are responsible for the remaining malaria burden.
Case definition	**Clinical case definition:** **Uncomplicated malaria** A patient with fever or history of fever within the past 48 hours (with or without other symptoms such as nausea, vomiting and diarrhoea, headache, back pain, chills, myalgia) in whom other obvious causes of fever have been excluded. **Severe malaria** A patient with symptoms as for uncomplicated malaria, plus drowsiness with extreme weakness and associated signs and symptoms related to organ failure (e.g. disorientation, loss of consciousness, convulsions, severe anaemia, jaundice, haemoglobinuria, spontaneous bleeding, pulmonary oedema and shock). **Confirmed case** Demonstration of malaria parasites in blood film by examining thick or thin smears, or by rapid diagnostic test for *P. falciparum.*
Mode of transmission	Vector-borne, through infective *Anopheles* mosquito bite. In Sudan, the primary malaria vectors are *An. arabiensis, An. gambiae* and *An. funestus.* Malaria may also be transmitted through blood transfusion of infected blood. Rarely, infants may contract malaria *in utero* through transplacental transfer of parasites, or during delivery.
Incubation	The incubation period for mosquito-transmitted infection is approximately 7–14 days for *P. falciparum,* 8–14 days for *P. vivax* and 7–30 days for *P. malariae.* However, malaria should be considered in all cases of unexplained fever that starts at any time between 1 week after the first possible exposure to malaria risk and 2 months (or even longer in rare cases) after the last possible exposure.
Period of communicability	Communicability is related to the presence of infective *Anopheles* mosquitoes and of infective gametocytes in the blood of patients. Untreated or insufficiently treated patients may be a source of mosquito infection for more than 3 years in *P. malariae* malaria, 1–2 years in *P. vivax* malaria and usually no longer than 1 year in *P. falciparum* malaria.

EPIDEMIOLOGY

Burden	**Number of malaria cases (2003):** **Reported:** 3 084 320 (including 1 085 953 laboratory-confirmed cases). Number of malaria deaths reported: 2 479(2003) **Estimated:** 7 500 000 clinical malaria cases annually. **Estimated malaria deaths per year:** 35 000.
Geographical distribution	Malaria risk is present throughout the country but is predominant in southern Sudan. Endemicity ranges from holoendemic in the south to hypoepidemic in the north, Epidemic-prone areas are confined to north/central Sudan.
Seasonality	Risk is year round-in the southern part of the country. In the North, malaria risk exists from July to November with a peak just after the rains.
Alert threshold	Any increase in the number of cases above what is expected for the time of the year in a defined area.
Recent epidemics	An epidemic that resulted from the absence of sufficient prevention measures was reported in White Nile state (2003).

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	The potential for epidemics can increase with the influx of non-immune populations moving from areas of no malaria/low transmission to highly endemic areas. Similarly, an influx of parasite carriers may trigger an epidemic in an epidemic-prone hypoendemic area.
Overcrowding	**Yes**	A consequence of increased population density and increased exposure to mosquito bites in temporary shelters.
Poor access to health services	**Yes**	• Delays in access to effective treatment increase the likelihood of severe disease and death. • Delays in access to effective treatment also increase the pool of malaria gametocyte carriers (the mature sexual stage of the parasite in humans that, once picked up in the blood-meal of a mosquito, develops into the infective stage for transmission to another human).
Food shortages	**No**	However, malnutrition increases vulnerability to severe malaria once infection has occurred. Case management also becomes more complicated, resulting in increased mortality.
Lack of safe water and poor sanitation	**No**	However, temporary surface-water bodies may increase breeding opportunities for the malaria vector.
Others	**Yes**	• Breakdown of control measures, and lack of preventive interventions (insecticide-treated materials such as bednets, sheeting, etc.) and indoor residual spraying of shelters with residual insecticide. • Changes in agricultural practice leading to extensive mosquito breeding sites. • Usually heavy rains and flooding.

Risk assessment conclusions	**Epidemiological situation** Malaria is the major health problem in Sudan, and the whole country is now considered endemic, with varying degrees. Malaria endemicity ranges from holo-endemic in the south to hypoendemic in the north with epidemic outbreaks. *P. falciparum* infection is overwhelmingly predominant (90%); *An. arabiensis* is the main vector; Country-wide 32% of outpatient attendance and 20% of all hospital deaths are due to malaria, which causes an estimated total of 7.5 million cases per year (Ministry of Health data, 1998). The situation is further aggravated by the spread of chloroquine-resistant *P. falciparum,* increasing insecticide resistance of vectors and inaccessibility of many areas, particularly in the south and west. It is hoped that the malaria situation will improve as a result of strong political commitment from the country towards that end, as well as the support generated from the RBM partnership. Training of health workers in case management and vector control is resulting in an improvement in malaria control activities. Epidemic preparedness and malaria information systems have markedly improved and helped in early detection and abortion of the expected malaria epidemic following the floods of 1998. Since 2002, a start has been made with the distribution of insecticide-treated nets (ITNs) were distributed throughout the country. The management system for malaria control in Sudan includes national malaria administration (NMCP) at central level and malaria units active in the majority of states. **Constraints** – Expansion of irrigation schemes and poor maintenance of drainage systems. – Lack of intersectoral and intrasectoral cooperation. – Massive population movement within the country. – Complex emergency situation in the south and west. – Inadequate surveillance systems. – Frequent turnover and emigration of technical staff to Arabian Gulf countries. **Priority actions adopted by the NMA** – Intensify malaria control in selected states (initiative in Gezira, Khartoum and Sinnar States). – Reduce malaria mortality in priority States. – Prevent epidemics in the country as a whole. – Support malaria control in complex emergency situation territories. – Prevent and control malaria in pregnancy through intermittent treatment in holoendemic stable transmission and irrigated areas. – Protect high-risk groups by insecticide-treated material. **National programme objectives** – To reduce incidence and morbidity of severe and complicated malaria cases, and prevent mortality. – To prevent, detect early and contain malaria epidemics.

PREVENTION AND CONTROL MEASURES

Case management

The new treatment protocol for malaria recommended by the Federal Ministry of Health and WHO/Sudan (Khartoum, June 2004) is summarized below:

Treatment of uncomplicated malaria

First-line treatment: (artesunate (AS) + sulfadoxine–pyrimethamine (SP).

Age (years)	Weight (kg)	Day 1		Day 2	Day 3
		SP (500 S + 25 P tablets)	**AS** (50 mg tablets)	**AS** (50 mg tablets)	**AS** (50 mg tablets)
<1	<10	½	½	½	½
1 – <7	10 <20	1	1	1	1
7–13	20 <40	2	2	2	2
14+	40+	3	4	4	4

Second-line treatment: (artemether 20 mg + lumefantrine 120 mg)=Coartem®

Age (years)	Weight (kg)	Day 1		Day 2		Day 3		Total no. of tablets
		0 time	8 hours	AM	PM	AM	PM	
	<10	**Use is not recommended; give oral quinine instead.**						
<1 – <3	10–14	1	1	1	1	1	1	6
3 – <7	15–24	2	2	2	2	2	2	12
7–11	25–34	3	3	3	3	3	3	18
12+	35+	4	4	4	4	4	4	24

Treatment of malaria in pregnancy

Weeks of amenorrhoea	Uncomplicated malaria	Severe malaria	Prevention
0–12	Quinine (Q)	Quinine (Q)	–
13–36	**1st opition**: Quinine or 3 days Q followed by SP **2nd Option** AS+ SP	Artemether or quinine	SP in two treatment doses, >1 month apart
37 until delivery	Quinine	Artemether or quinine	–
Puerperium	AS + SP	Artemether or quinine	–

	Treatment of severe malaria Quinine dihyrochloride, quinine hydrochloride or quinine sulfate 20 mg salt/kg (loading dose) diluted in 10ml/kg isotonic fluid by intravenous (IV) infusion over 4 hours. Eight hours after ***the start*** of the loading dose, give 10mg/kg over 4 hours. Repeat dose every 8 hours and shift to oral quinine as soon as the patient can tolerate oral medication. Quinine can be given intramuscularly (IM) in divided doses, with the same doses as above, diluted to 60mg/ml. **or** Quinine same as above (IV or IM) for at least 3 days and then shift to the first-line treatment (AS + SP). **or** Artemether IM 3.2mg/kg divided into 2 doses on the first day followed by 1.6mg/kg daily for the next 6 days. **or** Artemether IM 3.2/kg divided into 2 doses on the first day followed by 1.6mg/kg daily for at least 3 days and then shift to the first-line treatment (AS + SP). **Pre-referral treatment of severe malaria** Artesunate rectal capsules/suppositories, 10mg/kg, should be given as soon as possible once a diagnosis of severe malaria is made. If the rectal capsule is expelled within the first hour, another rectal capsule should be inserted immediately. A second dose can be repeated after 12 hours. THE PATIENT MUST BE TRANSPORTED WITHIN 24 HOURS. **or** Quinine 10 mg salt/kg IM in the standard dose. Repeat dose every 8 hours if referral is delayed. THE PATIENT MUST BE TRANSPORTED WITHIN 24 HOURS.
Prevention and Control	At present, malaria prevention measures in Sudan include distribution of insecticide treated mosquito nets, environmental management, distribution of Gambusia fish, larviciding and indoor residual spraying with pyrethroids. Free-of-charge drugs are provided in some endemic areas. **Chemoprophylaxis:** WHO recommends mefloquine, doxycycline or atovaquone/proguanil prophylaxis for expatriate staff traveling to Sudan. However according to new treatment guidelines, doxycline is not recommended. Chemoprophylaxis must be complemented by personal protection. It is not recommended on a population wide basis because it is extremely difficult to implement and assure compliance. Additionally, non-compliance to prophylaxis guidelines can accelerate the development of drug resistance. **Intermittent presumptive treatment (IPT)** at least twice during pregnancy (2nd and 3rd trimester) is advisable for pregnant women living in areas where transmission is high. National policy has recently changed to IPT in pregnancy with sulfadoxine–pyrimethamine, once in the 2nd trimester and again in the 3rd trimester. Vigorous **health education** at community level to improve rapid treatment-seeking behaviour for fever cases during the transmission season.

15. MEASLES

DESCRIPTION

Infectious agent	Measles virus (genus *Morbillivirus*, family Paramyxoviridae)
Case definition	**Clinical case definition:** Any person with: – Fever **and** – Maculopapular (i.e. non-vesicular) rash, **and** – Cough **or** coryza (i.e. runny nose) **or** conjunctivitis (i.e. red eyes); **or** Any person in whom a clinical health worker suspects measles infection. **Laboratory criteria:** Presence of measles-specific IgM antibodies. **Case classification:** Clinically confirmed: A case that meets the clinical case definition. Laboratory-confirmed (only for outbreak confirmation and during the outbreak prevention/elimination phase): – A case that meets the clinical case definition and is laboratory-confirmed. **or** – A case meeting clinical definition and epidemiologically linked by direct contact to a laboratory-confirmed case in which rash onset occurred 7–18 days earlier.
Mode of transmission	Airborne by droplet spread; or Direct contact with the nasal and throat secretions of infected persons or via objects (e.g. toys) that have been in close contact with an infected person.
Incubation	After infection there is an asymptomatic incubation period of 10–12 days, with a range from 7 to 18 days from exposure to the onset of fever.
Period of communicability	Measles is most infectious from 4 days before the rash until 1–2 days after rash onset.

EPIDEMIOLOGY

Burden	Number of cases reported:			
	2003:	4381 cases	**1997:**	350 cases
	2002:	4529 cases	**1990:**	14 075 cases
	2001:	4362 cases	**1980:**	50 168 cases
	2000:	2875 cases		
	1999:	3347 cases	(Data source: WHO–UNICEF estimates, 2004)	
	1998:	550 cases		
Geographical distribution	Measles is highly endemic throughout Sudan, and the expected number of measles cases is high.			
Seasonality	Higher incidence during the colder months. Some states experience two peaks (February–April and September–December).			

Alert threshold	One case in a state that has conducted a measles catch-up campaign (e.g Northern, River Nile, Red Sea and Kassala) must lead to an alert. Laboratory confirmation of all cases is not required. Only 5–10 cases from each outbreak need to be laboratory-confirmed.
Recent epidemics	It is reported that, in Sudan, one-third of all deaths in children aged under 3 years is due to measles. **October–November 2002**. 40 cases with 3 deaths were reported from Nimule (Magwe county, Eastern Equatoria). **October–November 2002**. 45 cases (no deaths) were reported from Gomjuer (Aweil West county, southern Bahr Al Ghazal). **September–October 2002**. 118 cases and 3 deaths were reported from Labone and Yei (Bahr Al Jebel). **July–August 2002**. 260 cases with 6 deaths were reported from areas of the Nuba mountains, including Jullud, Timen and Tima (Southern Kordofan). **March–April 2002**. 13 cases with 3 deaths were reported from Ruweng county (southern Sudan). **March–April 2001**. An outbreak involving 158 cases of measles was reported from Buoth and Mayoum (Upper Nile). **April–May 2001**. 14 cases were reported from Niemni (Upper Nile). **January–February 2001**: 101 cases and 26 deaths were reported from Nyimboli (Aweil West county, southern Bahr Al Ghazal). (Data source: WHO/Sudan)

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Importation of virus.
Overcrowding	**Yes**	Crowded conditions facilitate transmission.
Poor access to health services	**Yes**	Case-fatality rates can be reduced by effective case management, including the administration of vitamin A supplements.
Food shortages	**Yes**	Disease is more severe among children with malnutrition and vitamin A deficiency, and increases need for hospital care.
Lack of safe water and poor sanitation	**No**	
Others	**Yes**	Low immunization coverage in the area of origin of the refugees or internally displaced populations and/or in the host area. **MCV (measles-containing vaccine) coverage** **2001**: 80% (67% by WHO–UNICEF estimates) **2000:** 60% **1999:** 79% **1998:** 62% **1997:** 92% **1990:** 57% **1980:** NA (Data source: WHO/Sudan official country estimates)

Risk assessment conclusions	Routine immunization services have been hampered for several years due to civil unrest, leading to poor coverage rates, especially in the southern states. Measles is still common in various areas of the country, including the capital's area: the disease is the most common diagnosis among vaccine-preventable diseases in febrile children who present at the emergency hospitals in Khartoum.

PREVENTION AND CONTROL MEASURES

Introduction	Sudan has a routine immunization policy that requires a dose of single-antigen measles vaccine at 9 months of age (see Appendix 7*: Immunization schedule for Sudan)*. However, supplementary measles immunization activities are required in order to reduce the risk of a measles outbreak.
Routine Immunization	Immunize the population at risk as soon as possible. The priority is to **immunize children aged 6 months to 15 years**, regardless of vaccination status or history of disease. Expansion to older children is of lesser priority and should be based on evidence of high susceptibility among this age group. Children who are vaccinated against measles before 9 months of age must receive a second measles vaccination. This should be given as soon as possible after 9 months, with an interval of at least 1 month between doses. All children aged 6 months to 5 years should also receive prophylactic vitamin A supplementation. If there is evidence of clinical vitamin A deficiency in older age groups, treatment with vitamin A should be initiated as per WHO guidelines. To ensure safety of injection during immunization, auto-disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.
Outbreak response	Inform the health authorities immediately if one or more suspected cases are identified. Confirm the suspected outbreak, following WHO guidelines. Investigate suspected case: check if it fulfils the case definition, record date of onset, age and vaccination status. Confirm the diagnosis: collect blood specimen from 3–5 initial reported cases. Assess the extent of the outbreak and the population at risk. Implement outbreak response measures as follows: – Give priority to proper case management and immunization of groups at highest risk (e.g. children aged 6 months to 15 years) as soon as possible even in areas not yet affected but where the outbreak is likely to spread. – Promote social mobilization of parents in order to ensure that previously unvaccinated children aged from 6 months to 5 years are immunized. – The presence of several cases of measles in an emergency setting does not preclude a measles immunization campaign. Even among individuals who have already been exposed to, and are incubating, the natural virus, measles vaccine, if given within 3 days of exposure, may provide protection or modify the clinical severity of the illness. – Isolation is not indicated and children should not be withdrawn from feeding programmes.

Case management	**For uncomplicated cases:** — Give vitamin A immediately upon diagnosis and ensure the child receives a second dose the next day (can be given to parent to administer at home). — Advise the parent to treat the child at home (control fever and provide nutritional feeding). **For cases with non-severe eye, mouth or ear complications**: — Children can be treated at home. — Give vitamin A immediately upon diagnosis and ensure that the child receives a second dose the next day (can be given to parent to administer at home). — If pus draining from the eyes, clean eyes and treat with 1% tetracycline eye ointment. — If mouth ulcers, treat with gentian violet. — If pus draining from the ear, clean ear discharge and treat with antibiotics for 5 days (amoxicillin, first-line; or co-trimoxazole second-line-, as per national ARI policy and IMCI guidelines currently under development). — Treat malnutrition and diarrhoea, if present, with sufficient fluids and high-quality diet. **For cases with severe, complicated measles (any general danger signs*, clouding of cornea, deep or extensive mouth ulcers, pneumonia):** — Refer urgently to hospital. — Treat pneumonia with an appropriate antibiotic. — If clouding of the cornea or pus draining from the eye, clean eyes and apply 1% tetracycline eye ointment. — If the child has any eye signs indicating vitamin A deficiency (i.e. night blindness, Bitot spots, conjunctival and corneal dryness, corneal clouding or corneal ulceration), he or she should receive a third dose of vitamin A 2–4 weeks later. * Inability to drink or breastfeed, vomiting everything, convulsions, lethargy or unconsciousness.

16. MENINGOCOCCAL DISEASE (MENINGITIS AND SEPTICAEMIC FORM)

DESCRIPTION

Infectious agent	Bacterium: *Neisseria meningitidis* serogroups A, B, C, Y, W135
Case definition	**Clinical case definition:** An illness with sudden onset of fever (>38.5 °C rectal; >38.0 °C axillary) **and one or more** of the following: – neck stiffness – altered consciousness – other meningeal sign or petechial or purpural rash. In patients aged under one year, suspect meningitis when fever is accompanied by bulging fontanelle. **Laboratory criteria:** Positive CSF antigen detection, **or** Positive culture. **Case classification:** **Suspected:** a case that meets the clinical case definition above. **Probable:** a suspected case as defined above **and:** – Turbid CSF (with or without positive Gram-stain), **or** – Ongoing epidemic and epidemiological link to a confirmed case. **Confirmed:** a suspected or probable case with laboratory confirmation.
Mode of transmission	Direct contact with respiratory droplets.
Incubation	Incubation period varies between 2–10 days; most commonly 4 days.
Period of communicability	From the onset of symptoms until 24 hours after institution of therapy, but the most important sources of infection are asymptomatic carriers.

EPIDEMIOLOGY

Burden	**Cases and deaths of meningococcal meningitis reported to WHO/Sudan:** **2002**: no data reported **2001**: no data reported **2000:** 4031 cases, 328 deaths **1999:** 33 313 cases, 2410 deaths **1998:** 697 cases, 82 deaths **1997:** 297 cases **1996:** 340 cases
Geographical distribution	Epidemics occurred in Sudan between 1980–1999, affecting the following regions: Blue Nile, Darfur, Gezira, Kassala, Khartoum, Kordofan, Omdurman, Rumbek, Sinnar, White Nile.

Seasonality	Outbreaks tend to occur during the dry season (December–January).
Alert threshold[1]	**Population >30 000:** 5 cases per 100 000 inhabitants per week or a cluster of cases in an area. **Population <30 000:** 2 cases in 1 week or an increase in the number of cases compared with previous non-epidemic years. **Intervention:** (1) inform authorities; (2) investigate; (3) confirm; (4) treat cases; (5) strengthen surveillance; (6) prepare.
Epidemic threshold	**Population >30 000:** – 10 cases per 100 000 inhabitants per week if no epidemic for 3 years and vaccination coverage <80% or alert threshold crossed early in the dry season. – 15 cases per 100 000 inhabitants per week in other situations. **Population <30 000:** The population should be vaccinated if: – 5 cases in 1 week or – Doubling of the number of cases in a 3-week period **or** – For mass gatherings and displaced populations, 2 confirmed cases in 1 week. Other situations should be studied on a case-by-case basis. **Intervention:** 1. Mass vaccination. 2. Distribute appropriate antibiotics and case management protocols to health centres. 3. Treat according to epidemic protocol. 4. Maintain surveillance to track epidemic. 5. Inform the public. Current thresholds have been established from data in meningitis belt countries, which includes Sudan.

[1] Detecting meningococcal meningitis epidemics in highly-endemic African countries. Weekly Epidemiological Record, 2000, 38: 306–309.

Recent epidemics in the country	**2002**. As of 11 February, a total of 330 cases including 49 deaths were reported from Limun, Kauda and Hieman, in the Nuba mountains (Southern Kordofan). Serogroup A was confirmed. (CFR=14.8%). **January–March 2002**. As of 21 March 2003, 126 cases and 7 deaths were reported from Isoke (Torit county, Eastern Equatoria), and 104 cases and 14 deaths from Ikotos, in the same county **February–March 2002**. As of 15 March 2003, 8 cases and 1 death were reported from Padak (Boma county, Eastern Equatoria). **March 2001**. 42 admission cases and 2 deaths were reported from Jaibor (Keew county, Upper Nile). **February–March 2001**. As of 26 March, 19 cases and 1 death were reported from Chuil (Latjor county, Upper Nile). *N. meningitidis* serogroup A was identified. **February–March 2001**. As of 22 March, 67 cases and 13 deaths were reported from Paluer (Bor county, Upper Nile). **February 2001**. 117 cases and 1 death were reported from Narus (Eastern Equatoria). **2000**. A total of 2549 cases of meningococcal disease, of which 186 were fatal, were reported to the national health authorities between 1 January and 31 March 2000. Bahr Al Jebel State was the most affected, with 1437 cases (including 99 deaths) reported in the Juba city area. Other affected states included White Nile (197 cases, 15 deaths), Southern Kordofan and Sinnar (where incidence was lower). Epidemic response activities included vaccination of a total of 70 000 people in early March. **1998–1999**. An outbreak of meningococcal meningitis was reported in the Northern Darfur region. An increase in the number of cases had already been observed in December 1998. As far as May 1999, about 22 000 cases of meningococcal disease had been notified from 19 of the 26 states of Sudan, of which 1600 had died. More than 10 million doses of meningococcal vaccine were distributed for mass vaccination campaigns. In 1999, a total of 33 313 cases and 2410 deaths were reported from Sudan to WHO. **1988**. Following the return of pilgrims from Mecca (Hajj) in August 1987, many countries in EMR faced an unusual spread of meningococcal infection. In Sudan, the 1987 introduction developed into epidemic spread in the meningitis season of 1988, when 32 016 cases of meningococcal disease were reported to WHO.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Travel, migration and displacement facilitate the circulation of virulent strains within a country or from country to country.
Overcrowding	**Yes**	High density of susceptible people is an important risk factor for outbreaks. Internally displaced populations and refugee camps, crowding because of cattle or fishing-related activities, military camps and schools facilitate spread of the disease.
Poor access to health services	**Yes**	Case identification is crucial to rapidly implement control measures. The case-fatality rate without treatment is very high .
Food shortages	**No**	
Lack of safe water and poor sanitation	**No**	
Others	**No**	Concurrent infections: upper respiratory tract infections may contribute to some meningococcal outbreaks. Dry and windy/dusty conditions increase transmission of the disease.

Risk assessment conclusions	Central and southern Sudan are included in the African meningitis belt, which extends from Ethiopia in the east to Senegal in the west, mainly within the annual rainfall range of 300–1100 mm. In this area, sporadic infections occur in seasonal, annual cycles; large-scale epidemics occur at greater intervals with irregular patterns, usually beginning during the dry season (December–February), and sometimes lasting for more than a year. Since the early 1990s, Sudan has been practising preventive vaccination, mainly directed to high-risk groups. However, this was not sufficient to prevent the 1999 epidemics due to mobility of populations. There is a high risk of epidemics in overcrowded camps.

PREVENTION AND CONTROL MEASURES

Case management

Meningococcal disease (either meningitis or septicaemia) is potentially fatal and should always be viewed as a medical emergency.

NON-EPIDEMIC CONDITIONS:

- Admission to a hospital or health centre is necessary for diagnosis (lumbar puncture and CSF examination). As soon as mengitis is suspected, a lumbar puncture must be done before starting antibiotic treatment
- As infectivity of patients is moderate and disappears quickly following antimicrobial treatment, isolation of the patient is **not** necessary.
- Antimicrobic treatment must be instituted as soon as possible after lumbar puncture (without waiting for laboratory results), and should be combined with supportive treatment.

Initial antimicrobial therapy should be effective against the three major causes of bacterial meningitis **until bacteriological results are available:**

AGE GROUP	PROBABLE PATHOGENS	ANTIBIOTIC THERAPY	
		FIRST CHOICE	ALTERNATIVE
Adults and children < 5years	S. pneumoniae	Benzylpenicillin	Ampicillin or amoxicillin Chloramphenicol Ceftriaxone or cefotaxime
Children 1 month - 5 years	H. Influenza S. pneumoniae N. meningitidis	Ampicillin or amoxicillin[a]	Chloramphenicol Ceftriaxone or cefotaxime
Neonates	Gram-negative bacteria Group B streptococci Listeria	Ampicillin and gentamicin	Ceftriaxone or cefotaxime[b]

[a] If *H. influenzae* is highly resistant to ampicillin, chloramphenicol should be given with ampicillin.
[b] No effect on listeria.

Once diagnosis of meningococcal disease has been established, many antimicrobials can be used:

- either *penicillin* or *ampicillin* is the drug of choice.
- *Chloramphenicol* is a good and inexpensive alternative.
- The third-generation cefalosporins, *ceftriaxone* and *cefotaxime*, are excellent alternatives but are considerably more expensive.
- A 7-day course is still the general rule for the treatment of meningococcal disease (except in the neonatal period where a 14-day course is given).

	– The long-acting (oily) form of chloramphenicol has also been shown to be effective. The Sudan Federal Ministry of Health recommends injectable oily choloramphenicol and *benzylpenicillin*. **EPIDEMIC CONDITIONS:** During epidemics of confirmed meningococcal disease, case management needs to be simplified to permit the health system to respond to rapidly increasing numbers of cases. • Diagnosis: as the flood of patients could make the routine use of lumbar puncture to confirm meningitis impossible, every suspected case of meningitis should be considered and treated as one of meningococcal meningitis. • Treatment: simplified treatment protocols are appropriate: long-acting oily chloramphenicol (100 mg/kg up to 3 g in a single dose) IM is the drug of choice for all age groups, particularly in areas with limited health facilities. For patients who do not improve rapidly, an additional dose of the same antimicrobial is recommended 48 hours later.
Prevention	**NON-EPIDEMIC CONDITIONS:** • Vaccination: to prevent secondary cases around a sporadic case of meningococcal disease, vaccine can be used for close contacts of patients with meningococcal disease due to serogroup A, C or W135. • Chemoprophylaxis: the aim of chemoprophylaxis is to prevent secondary cases by eliminating nasopharyngeal carriage. To be effective in preventing secondary cases, chemoprophylaxis must be initiated as soon as possible (i.e. not later than 48 hours after diagnosis of the case). Its use should be restricted to close contacts of a case, which are defined as: – Household members (i.e. persons sleeping in the same dwelling as the case); – Institutional contacts (i.e. persons who share sleeping quarters (i.e. roommates in boarding schools; persons sharing a barracks in military camps); nursery school or childcare centre contacts (i.e. children and teachers who share a classroom with the case); – Other persons who have had contact with the patient's oral secretions through kissing or sharing of food and beverages.

	EPIDEMIC CONDITIONS • **Vaccination**: a mass vaccination campaign, if appropriately carried out, can halt an epidemic of meningococcal disease. Laboratory diagnosis and confirmation of epidemic serogroups will guide the type of vaccine needed, either meningococcal polysaccharide bivalent A/C (if serogroup A or C is confirmed as the epidemic serogroup), or meningococcal polysaccharide tetravalent vaccine A/C/Y/W135 (if serogroup Y or W135 is confirmed). Vaccination should be targeted to areas where the epidemic threshold is reached. Camp population: Following confirmation (serogroup identified) of two cases, mass vaccination is recommended if the serogroup/s identified is/are included in either the bivalent (A/C) or tetravalent (A/C/Y/W135) vaccine. At-risk populations (e.g. aged 2–30 years) should be given priority. – General population: If an outbreak is suspected, vaccination should only be considered after careful investigation (including confirmation and serogroup identification) and the assessment of the population group at highest risk. • **Chemoprophylaxis**: chemoprophylaxis of contacts of meningitis patients is NOT warranted during an epidemic for several reasons. In small clusters or outbreaks among closed populations (e.g. extended household, boarding schools), chemoprophylaxis may still be appropriate.

17. ONCHOCERCIASIS (RIVER BLINDNESS)

DESCRIPTION

Infectious agent	*Onchocerca volvulus*, a filarial worm belonging to the class Nematoda.
Case definition	**Clinical description** Persons suffering from onchocerciasis may experience: **(A) Skin lesions:** dermal changes are secondary to tissue reaction to motile larvae as they migrate subcutaneously, or to their destruction in the skin. – **Itching:** the pruritus of onchocerciasis is the most severe and intractable that is known. In lightly infected persons, this may persist as the only symptom. – **Rashes:** the rash usually consists of many raised papules, which are due to microabscess formation, and may disappear within a few days or may spread. *Sowda*, from the Arabic for black or dark, is an intensely pruritic eruption usually limited to one limb and including oedema, hyperpigmented papules and regional lymphadenopathy. It is common in Yemen, but can also be found in Sudan. – **Depigmentation of the skin:** areas of depigmentation over the anterior shin with islands of normally pigmented skin, commonly called "leopard skin", are found in advanced dermatitis. – **Subcutaneous nodules:** these are 0.5–3.0 cm asymptomatic subcutaneous granulomas resulting from a tissue reaction around adult worms. They occur most often over bony prominences: in Africa the nodules are often located over the hips and lower limbs. – **Lymphadenopathy:** it is frequently found in inguinal and femoral areas, and can result in "hanging groin" (especially when associated with skin atrophy and loss of elasticity) and elephantiasis of the genitalia. **(B) Eye lesions:** ocular onchocerciasis is related to the presence of live or dead microfilariae. Involvement of all tissues of the eye has been described, and many changes in both anterior and posterior segments of the eye can occur. The more serious lesions lead to serious visual impairment, including blindness. **(C) General debilitation:** onchocerciasis has also been associated with weight loss and musculoskeletal pain. **Clinical case definition** In an endemic area, a person with fibrous nodules in subcutaneous tissues. These must be distinguished from lymph nodes or ganglia. **Laboratory criteria** Presence of one or more of the following: – Microfilariae in skin snips taken from the iliac crest (Africa) or scapula (Americas) – Adult worms in excised nodules – Typical ocular manifestations, such as slit-lamp observations of microfilariae in the cornea, the anterior chamber, or the vitreous body – Serology (especially for non-indigenous persons) **Case classification** – Suspected: A case that meets the clinical case definition. – Probable: Not applicable. – Confirmed: A suspected case that is laboratory-confirmed.

Mode of transmission	Vector-borne, by the bite of infected female blackflies belonging to the genus *Simulium* (mainly *S. damnosum* complex). Larvae of the vector for onchocerciasis lay their eggs in the water of fast-flowing rivers – hence the name "river blindness". Microfilariae are ingested by a blackfly feeding on an infected person, and penetrate thoracic muscles of the fly. Here, a few of them develop into infective larvae after several days, migrate to the cephalic capsule, are liberated onto the skin and enter the bite wound during a subsequent blood-meal. Infective larvae develop into adult parasites in the human body, where adult forms of *O. volvulus* can live for up to 14–15 years and are often found encased in fibrous subcutaneous nodules. Each adult female produces millions of microfilariae that migrate under the skin and through the eyes, giving rise to a variety of dermal and ocular symptoms. Humans are the only reservoir. Other *Onchocerca* species found in animals cannot infect humans but may occur together with *O. volvulus* in the insect vector.
Incubation	Larvae take at least 6–12 months to become adult worms. Adult worms are usually innocuous, apart from the production of the subcutaneous nodules (these can develop as early as 1 year after infection). The main pathologic sequelae of *O. volvulus* infection are caused by the microfilariae in skin and ocular tissue, where they can be found after a period of 7–34 months. Microfilariae are found in the skin usually only 1 year or more after the time of the infective bite.
Period of communicability	**Human → blackfly** Infected individuals can infect blackflies as long as living microfilariae occur in their skin. Microfilariae are continuously produced by adult female worms (about 700 per day), and can be found in the skin after a prepatent period of 7–34 months following introduction of infective larvae. They may persist for up to 2 years after the death of the adult worms. **Blackfly → human** Blackfly vectors become infective (i.e. able to transmit infective larvae) 7–9 days after the blood-meal.

EPIDEMIOLOGY

Burden	An estimated 2 million persons are at risk of onchocerciasis in Sudan, with 10 000 cases of onchocerciasis-related blindness. **Southern focus:** in Western Bahr Al Ghazal, more than 80% of subjects in some villages had palpable nodules in 1998. **Northern focus:** a rapid epidemiological assessment (REA) in 1995 revealed that 16% of local inhabitants had palpable nodules. Skin-snip positivity reached 33.6%. **Eastern focus:** skin-snip positivity may reach 50% in some villages, but nodule rates were low (1998). **Western focus:** an REA in 1996 revealed that 22% of subjects in this area had palpable nodules; 28% had onchocercal skin lesions or itching.

Geographical distribution	Infection with *O. volvulus is* known to occur in four main regions, known as the southern, northern, eastern and western foci. **Southern focus:** this is the largest focus and includes all the southern states from Western Bahr Al Ghazal in the west to Upper Nile in the east. The rate of infection is highest in the south-west, in Western Bahr Al Ghazal State, especially among communities living along the Jur river (Nahr el Jur) and its tributaries. **Northern focus:** the rocky ground and river bed between the fourth and fifth Nile cataracts create breeding sites for *S. damnosum.* The river level reaches its peak in late summer (August–September), and ideal conditions for *Simulium* breeding occur when the water recedes and rocks and vegetation emerge, creating surface turbulence. This focus is located in the Abu Hamad area of the Nubian desert in Northern State. This is the most northerly focus in Africa, and probably in the world. **Eastern focus:** this focus is located in Gedaref State, along the upper Atbara river, close to the Ethiopian border. **Western focus:** this focus is contiguous to the southern one and includes the communities living along the Umbellasha, Adda and Bahr El Arab River in Southern Darfur. This is a savannah region where seasonal rainfalls (May–September) give rise to fast-flowing rivers that, during the dry months, become parched or stagnant.
Seasonality	**Southern focus:** transmission from July to October. **Northern focus:** transmission between November and January. **Eastern focus:** transmission from July to October. **Western focus:** transmission from May to September.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Infections in the Bahr El Arab area (western focus) are probably the result of individuals from the southern focus migrating northwards during the civil war in the 1950s.
Overcrowding	**No**	
Poor access to health services	**Yes**	Community-directed treatment with ivermectin (CDTI) is an effective tool for transmission control. People can infect flies as long as living microfilariae occur in their skin.
Food shortages	**No**	
Lack of safe water and poor sanitation	**No**	
Others	**No**	

Risk assessment conclusions	"Craw-craw" (itchy skin due to onchocerciasis) was reported for the first time in Sudan in 1908 from Bahr Al Ghazal. The Global 2000 River Blindness Program of The Carter Center estimates the crude ultimate treatment goal for Sudan (total number of people in need of treatment) at 743 230. Of the several endemic foci, the southern focus is the most significant and is characterized by high prevalence of blinding onchocerciasis. Some of the highest rates of blindness due to onchocerciasis in the world occur in south-west Sudan. The cost per treatment in 2001 was considerably above that recommended by APOC, and calculated at US$ 0.74. The high cost underscores the principle that distribution in conflict areas will be more expensive. In general, communities are committed to the distribution of ivermectin. The onchocerciasis control programme is viewed at the highest governmental levels as an example of a successful health delivery system and is well integrated into the Sudanese primary health care system. Among the constraints encountered in Sudan: – Accessibility problems due to the civil unrest, flood, famine, drought and mass population displacement (in southern Sudan). – Continuous reshaping of the population and the community-directed drug distributors. – Impaired treatment activities in areas devoid of any health infrastructure, or in areas where the primary health care system is not operational. – Coendemicity for *O. volvulus* and *Loa loa* in some areas of south-western Sudan. Guidelines for *Loa loa* coendemic areas still need to be implemented. It seems likely that there has been a decrease in the prevalence of infection in the past one or two decades in the western focus. This reduction may be the result of mass population migration and reduced vector density following a period of drought in 1987, and also of annual ivermectin treatment. The prevalence of infection appeared to be reduced also in the northern focus in 1995 when compared with 1985, thanks to the five annual rounds of ivermectin treatment carried out in this area.

PREVENTION AND CONTROL MEASURES

Case management	Administration of ivermectin once a year over a period of at least 15–20 years reduces infection to insignificant levels and prevents the appearance of clinical manifestations. The recommended dosage is equivalent to 150 µg/kg of body weight (in practice, dosage is according to height, using 1–4 tablets of 3 mg). Established clinical manifestations are also treated by ivermectin. Treatment with ivermectin is contraindicated in: – Children under 5 years (age), less than 15 kg (weight), or less than 90 cm (height). – Pregnant women – Lactating mothers of infants less than 1 week old – Severely ill persons ***NB:*** *Ivermectin should be used with extreme caution in areas coendemic for* Loa loa*.*
Epidemic control	Recrudescence of transmission may occur and can be managed by the mass administration of ivermectin where programmes can maintain good treatment coverage.

Prevention	• **Vector control** Destruction of *Simulium* larvae by application of insecticides such as temephos (Abate®) through aerial spraying to breeding sites in fast-flowing rivers, in order to interrupt the cycle of disease transmission. Once the cycle has been interrupted for 14–15 years, the reservoir of adult worms dies out in the human population, thus eliminating the source of the disease. This was the basic strategy of the Onchocerciasis Control Programme (OCP) in west Africa. In the west African savannah zone, onchocerciasis was a severely blinding disease. It was also responsible for the depopulation of fertile river valleys in OCP countries and was therefore a major impediment to economic development. For these reasons, the **large-scale vector control operations** of the OCP – based on the aerial application of insecticides and aiming at the virtual elimination of the disease – were considered economically justified. OCP has been successful in eliminating onchocerciasis as a public health and socioeconomic problem. The African Programme for Onchocerciasis Control (APOC), which includes Sudan and other 18 African countries, uses **focal vector eradication** as a control option. This implies that the whole focus is covered at once, resulting in the total eradication of the vector over a very short timescale. • **Community-directed treatment with ivermectin (CDTI):** ***Course****: Once-a-year administration of ivermectin 150* µg/*kg of body weight* The introduction of ivermectin in 1987 provided for the first time a feasible chemotherapy regimen for large-scale treatment of onchocerciasis. Ivermectin is an effective microfilaricide that greatly reduces the numbers of skin microfilariae to low levels for up to a year, thus: (1) alleviating many symptoms: since the microfilariae cause the severe morbidity of onchocerciasis, ivermectin treatment is an effective tool for morbidity control, able to prevent the development of ocular lesions and blindness; (2) making the recipient less infective for the vector: ivermectin is an effective tool for onchocerciasis transmission control. Ivermectin treatment greatly reduces transmission of the parasite but does not halt it within the period of a decade or more, and the adult worm may live for as long as 14–15 years. Annual large-scale treatment will therefore have to continue for a very long time. Current predictions with a simulation model indicate that annual treatment at the current level of coverage may have to continue for at least two decades. The main challenge facing ivermectin-based control is therefore to develop and implement simple methods of ivermectin delivery that can be sustained by the communities themselves. The current APOC strategy consists of house to-house distribution or at central meeting points in villages. CDTI is the main strategy adopted by APOC. In the 19 countries included in this programme, onchocerciasis remains a major cause of blindness but does not appear to be the cause of major depopulation of fertile lands. Partly for this reason, large-scale vector control operations are not likely to be as cost-effective as they have been in the OCP area.

18. PERTUSSIS (WHOOPING COUGH)

DESCRIPTION

Infectious agent	*Bordetella pertussis*, the pertussis bacillus.
Case definition	**Clinical description:** The initial stage, the **catarrhal stage**, is characterized by the insidious onset of coryza (runny nose), sneezing, low-grade fever and a mild, occasional cough, similar to the commom cold. The cough gradually becomes more severe and irritating, and after 1–2 weeks the second stage, or **paroxysmal stage**, begins. The patient has bursts, or paroxysms, of numerous, rapid coughs, apparently due to difficulty in expelling thick mucus from the tracheobronchial tree. At the end of the paroxysm, a long inspiratory effort is usually accompanied by a characteristic whoop. In younger infants, periods of apnoea may follow the coughing spasms, and the patient may become cyanotic (turn blue). Pneumonia is a relatively common complication (reported 21.7% of cases in developed countries); otitis, haemorrhages (subconjunctival petechiae and epistaxis), convulsions, encephalopathies and death occur more rarely). The disease lasts 4–8 weeks. Complications are more frequent and severe in younger infants. In developed countries, the case-fatality rate among infants aged less than 1 month has been reported to be around 1%. Older persons (adolescent and adults) and those partially protected by the vaccine may become infected with *B. pertussis* but usually have milder disease. In the **convalescent stage**, recovery is gradual. The cough becomes less paroxysmal and disappears over 2–3 weeks. However, paroxysms often recur with subsequent respiratory infections for many months after the onset of pertussis. Fever is generally minimal throughout the course of pertussis. **Clinical case definition:** A case diagnosed as pertussis by a physician, or A person with a cough lasting at least 2 weeks **with at least one** of the following symptoms: – Paroxysms (i.e. fits) of coughing – Inspiratory "whooping" – Post-tussive vomiting (i.e. vomiting immediately after coughing). **Laboratory criteria:** – Isolation of *Bordetella pertussis*, or – Detection of genomic sequences by polymerase chain reaction (PCR) – Positive paired serology. **Case classification:** **Clinical case:** A case that meets the clinical case definition. **Confirmed case:** A clinical case that is laboratory-confirmed.
Mode of transmission	Primarily by direct contact with discharges from respiratory mucous membranes of infected persons via the airborne route. Humans are the only hosts. Although the disease may be milder in older persons, these infected persons may transmit the disease to other susceptible persons, including non-immunized or under-immunized infants. An adult is often found to be the first case in a household with multiple pertussis cases.

Incubation	The incubation period usually lasts 7–10 days; rarely more than 14 days.
Period of communicability	Pertussis is highly communicable in the early catarrhal stage. Communicability gradually decreases after onset of paroxysmal cough. Untreated patients may be contagious for up to 3 weeks after onset of paroxysmal cough without treatment or for up to 5 days after onset of treatment.

EPIDEMIOLOGY

Burden	**Number of cases reported** **Year Cases** 2003 232 2002 213 2001 645 2000 80 1999 51 1998 169 1997 418 1990: 566 1980 28 631 (Source: WHO–UNICEF data, 2004)
Geographical distribution	No data available.
Seasonality	Pertussis has no distinct seasonal pattern, but activity may increase in the summer and autumn.
Alert threshold	
Recent epidemics in the country	**2002 December**: 5 cases (no deaths) were reported from Akob Payam (Tonj county, Lakes state). **2002 October–December**: 127 cases with 2 deaths were reported from Akon (Gogrial county, Western Bahr Al Ghazal). **2002 August–September**: 68 cases and 5 deaths were reported from several villages in Oriny and Shilluk counties (Upper Nile state). All cases, except 5, were children aged <5 years.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Importation and spread of *B. pertussis.*
Overcrowding	**Yes**	Crowded conditions facilitate transmission. The disease is usually introduced into a household by an older sibling or a parent.
Poor access to health services	**Yes**	No access to routine immunization services. Susceptibility of non-immunized individuals is universal, and vaccination is the mainstay of pertussis control.
Food shortages	**No**	

Lack of safe water and poor sanitation	**No**	
Others	**Yes**	Low DTP3 coverage (<80%). **DTP3 coverage:** **2001:** 71% (46% by WHO–UNICEF estimates) **2000:** 65% **1999:** 79% **1998:** 70% **1997:** 79% **1990:** 62% **1980:** 1 % (Data source: WHO/Sudan official country estimates)
Risk assessment conclusions	Yearly fluctuations in the number of reported cases reflect a weak surveillance system. Pertussis is a potential problem if introduced into crowded conditions with many non-immunized children. It is highly contagious. Children aged under 1 year and pregnant women are at greatest risk.	

PREVENTION AND CONTROL MEASURES

Case management	• Erythromycin or erythromycin estolate or – in case of allergies to erythromycin – trimethoprim–sulfamethoxazole (contraindicated during pregnancy) should be administered for 7–14 days to all **cases** and close **contacts** of persons with pertussis, regardless of age and vaccination status. Drug administration both (1) modifies the course of illness (if initiated early) and (2) eradicates the organism from secretions, thereby decreasing communicability. • Symptomatic treatment and supportive case-management. It is important that vaccination coverage is improved. Health workers should be trained to recognize and treat cases and contacts as indicated below.
Immunization	Vaccination is the most effective way to control pertussis. Active primary immunization against *B. pertussis* infection with the whole-cell vaccine (wP) is recommended in association with the administration of diphtheria and tetanus toxoids (DTP). No single-antigen pertussis vaccine is available. Although the use of acellular vaccines is less commonly associated with adverse reactions, price considerations affect their use, and wP vaccines are the vaccines of choice for most countries, including Sudan. In general, pertussis vaccine (wP) is not given to persons aged 7 years or older, since local reactions (convulsions, collapse, high temperature) may be increased in older children and adults. The efficacy of the vaccine in children who have received at least 3 doses is estimated to be 80%: protection is greater against severe disease and begins to wane after about 3 years.

Epidemic control	The highly contagious nature of the disease leads to large numbers of secondary cases among non-immune contacts. Prophylactic antibiotic treatment (with erythromycin) in the early incubation period may prevent disease, but difficulties of early diagnosis, the costs involved and concerns related to the occurrence of drug resistance all limit prophylactic treatment to selected individual cases. Priority must be given to: • Protecting children aged under 1 year and pregnant females in the last 3 weeks of pregnancy because of the risk of transmission to the newborn. • Stopping infection among household members, particularly if the household includes children aged under 1 year and pregnant women in the last 3 weeks of pregnancy. The strategy relies on chemoprophylaxis of contacts within a maximum delay of 14 days following the first contact with the index case. Index cases must avoid contact with childcare centres, schools and other places where susceptible individuals are grouped for up to 5 days after starting treatment, or for up to 3 weeks after onset of paroxysmal cough, or until the end of cough, whichever comes first. All contact cases must have their immunization status verified and brought up to date.

19. POLIOMYELITIS

DESCRIPTION

Infectious agent	Poliovirus (Enterovirus group): types 1, 2, 3.
Case definition and classification	**Clinical description:** All three types of wild poliovirus may cause paralysis, although most infections (at least 95%) remain asymptomatic. Most symptomatic cases report a nonspecific febrile illness lasting a few days, corresponding to the **viraemic** phase of the disease. In a few cases, fever can be followed by the abrupt onset of **meningitic and neuromuscular** symptoms such as stiffness in the neck and pain in the limbs. Initial symptoms may also include fatigue, headaches, vomiting, constipation (or, less commonly, diarrhoea). In a very small percentage of cases (≤1 of 100 infected susceptible persons), this is followed by gradual onset (2–4 days) of flaccid paralysis. **Paralytic** disease usually affects the lower limbs and is typically asymmetric and more severe proximally. Bulbar (brainstem) paralysis may also occasionally occur, leading to respiratory muscle involvement and death unless artificial respiration can be applied. The mortality from paralytic poliomyelitis is 2–10%, mainly as a result of bulbar involvement and/or respiratory failure. • Risk factors for paralytic disease are a large inoculum of virus, increasing age, pregnancy, recent tonsillectomy, strenuous exercise and intramuscular injections during the incubation period. • After the acute illness there is often a degree of recovery of muscle function; 80% of eventual recovery occurs within 6 months, although recovery of muscle function may continue for up to 2 years. • After many years of stable neurological impairment, new neuromuscular symptoms (weakness, pain and fatigue) develop (post-polio syndrome) in 25–40% of patients. **Clinical case definition:** • Acute flaccid paralysis (AFP) in a child aged <15 years, including Guillain - Barré syndrome*; **or** • Any paralytic illness in a person of any age when poliomyelitis is suspected. * For practical reasons, Guillain–Barré syndrome is considered as poliomyelitis until proven otherwise. **Case classification:** • Suspected: A case that meets the clinical case definition. • Confirmed: AFP with laboratory-confirmed wild poliovirus in stool sample. • Polio-compatible: AFP clinically compatible with poliomyelitis, but without adequate virological investigation.
Mode of transmission	Poliovirus is highly communicable. Transmission is primarily from person to person via the faecal–oral route.
Incubation	The time between infection and onset of paralysis is 4–30 days.
Period of communicability	From 36 hours after infection, for 4–6 weeks.

EPIDEMIOLOGY

Burden	**Number of confirmed wild polio virus cases reported:** **2004:** 1 **2003:** 0 **2002:** 0 **2001:** 1 **2000:** 4 **1999:** 10 **1998:** 8 **1997:** 13 **1996:** 6 (Source: WHO/Polio Eradication initiative, 2004)
Geographical distribution	
Seasonality	
Alert threshold	Any AFP case must be notified and investigated.
Recent epidemics in the country	**2004***.* A wild poliovirus case genetically linked to the genotypes circulating in Northern Nigeria was confirmed during the humanitarian crisis in Darfur. **1993***.* The earliest cases of a large outbreak were reported from Darfur State in May, reaching a peak in July, and spreading to eight of nine states *(since then administrative division of the country has changed)* by December 1993. The highest reported incidence was from Darfur and Equatoria, where infant coverage for the third dose of oral poliomyelitis vaccine was reported to be 29% and 23% respectively in 1993. A total of 252 cases were confirmed as poliomyelitis according to WHO criteria; 85% were aged under 5 years, 53% were male and 57% were from urban areas. Only 26% of cases had received the third dose of OPV, suggesting that the outbreak was largely due to an accumulation of susceptible children and was accelerated by low immunization coverage.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Importation of virus.
Overcrowding	**Yes**	Very important in facilitating transmission.
Poor access to health services	**Yes**	No access to routine immunization services. Risk of undetected poliovirus circulation.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	Generally poor sanitation.
Others	**Yes**	Low OPV3 coverage (<80%). **2001:** 71% (47% by WHO–UNICEF estimates) **2000:** 65% **1999:** 78% **1998***:* 68% **1997***:* 78% **1990:** 62% **1980:** 1% (Data source: WHO/Sudan official country estimates)

PREVENTION AND CONTROL MEASURES

Risk assessment conclusions	The civil war in southern Sudan has hampered routine immunization services for more than 20 years. However, high-quality supplementary immunization activities have been implemented in the country, especially in recent years. The recently confirmed case in Darfur emphasizes the need to maintain vigilant AFP surveillance and high OPV immunization coverage. Sub-NIDs targeting children aged <5 years in southern Sudan held in October and November 2002 reached an unprecedented number of children. During the first round, 755 877 children were vaccinated (compared with 539 845 in 2001). In the second round, 1 108 316 children were vaccinated. Unimpeded access throughout southern Sudan allowed the polio programme to reach some areas for the first time. Management of polio eradication efforts has been split between government-controlled zones and rebel-dominated territories. The last reported wild poliovirus-positive case in Sudan occurred in May 2004. Supplementary immunization activities began in 1994 in Sudan.
Case management	Management of the acute phase of paralytic poliomyelitis is supportive and symptomatic: — Bed rest. — Close monitoring of respiration: respiratory support in case of respiratory failure or pooling of pharyngeal secretions. — Moist hot-packs for muscle pain and spasms. — Passive physical therapy to stimulate muscles and prevent contractures. — Anti-spasmodic drugs. — Frequent turning to prevent bedsores. If hospitalization is required, the patient should be isolated. Disinfection of discharges, faeces and soiled articles, and immediate reporting of further cases are essential.

Immunization	Two types of poliovirus vaccine are available: • Oral poliovirus vaccine (OPV): OPV is an orally administered vaccine that includes live attenuated strains of all three virus types. It is easily administered by health workers or volunteers, induces a good humoral (antibody) and mucosal (intestine) immune response and is four times cheaper than inactivated poliovirus vaccine (IPV). OPV is the only vaccine of choice for poliomyelitis eradication because it achieves much better mucosal immunity than IPV while limiting the dissemination of wild poliovirus in the community. • Inactivated poliovirus vaccine (IPV): IPV can be given only by intramuscular injection and requires trained health workers. It elicits an excellent antibody response but only minimal intestinal mucosal response; it is much more expensive than IPV. Sudan has a **routine immunization policy** that requires 4 doses of OPV (see Appendix 7: *Immunization schedule for Sudan*). However, **supplementary immunization activities** are also conducted in the country in order to maximize immunization coverage: these consist of national immunization days (NIDs), sub-NIDs (mass campaigns similar to NIDs but confined to a smaller geographical area), and mop-up campaigns, during which 2 OPV doses are given at an interval of 1 month to all children aged under 5 years, preferably during the low transmission season for enteroviruses (the cooler season). Supplementary immunization activities in Sudan: NIDs started in 1994 for polio eradication activity: - 11 national campaigns have been conducted, with two rounds for each. - 5 sub-NIDs in selected high-risk areas conducted two rounds for each. - MNT (maternal and neonatal tetanus elimination campaigns were conducted in high-risk localities (28) from 2000–2003. **Among displaced populations, all children aged 0–59 months should be vaccinated on arrival.** **Any AFP case must be notified and investigated.**
Epidemic Control	In case of a suspected outbreak: **Investigation** – Clinical and epidemiological investigation. – Rapid virological investigation (2 stool samples taken within 14 days of onset of paralysis should be sent to a WHO-accredited laboratory). Outbreak confirmation will be based on the isolation of wild poliovirus. **Intervention** A house-to-house mop-up campaign with OPV should be conducted in a wide geographical area (at least province involved and relevant neighbours) if no NIDs or sub-NIDs are planned to cover the area within the next 3 months. If NIDs or sub-NIDs are planned, focus should be set on ensuring that high-quality immunization activities are implemented in the area of the outbreak and adjacent districts. Surveillance should be enhanced through intensive monitoring of all reporting units, ensuring active surveillance and zero reporting, extensive retrospective record reviews and active case-finding in surrounding areas.

20. RABIES

DESCRIPTION

Infectious agent	Rabies virus, a Rhabdovirus of the genus *Lyssavirus*.
Case definition	**Clinical description** • Paresis or paralysis, delirium, convulsions. • Without medical attention, death in about 6 days, usually due to respiratory paralysis. **Clinical case definition** An acute neurological syndrome (encephalitis) dominated by forms of hyperactivity (furious rabies) or paralytic syndrome (dumb rabies) that progresses towards coma and death, usually from respiratory failure, within 7–10 days after the first symptom. **Laboratory criteria** One or more of the following: – Detection of rabies viral antigens by direct fluorescent antibody (FA) in clinical specimens, preferably brain tissue (collected post mortem). – Detection by FA on skin or corneal smear (collected antemortem). – FA positive after inoculation of brain tissue, saliva or CSF in cell culture, in mice or in suckling mice. – Detectable rabies-neutralizing antibody titre in the CSF of an unvaccinated person. – Identification of viral antigens by PCR on fixed tissue collected post mortem or in a clinical specimen (brain tissue or skin, saliva or urine). – Isolation of rabies virus from clinical specimens and confirmation of rabies viral antigens. **Case classification** **Human rabies:** – **Suspected:** A case that is compatible with the clinical case definition. – **Probable:** A suspected case plus history of contact with a suspected rabid animal. – **Confirmed:** A suspected case that is laboratory-confirmed. **Human exposure to rabies**: – **Possibly exposed:** A person who had close contact (usually a bite or a scratch) with a rabies-susceptible animal in (or originating from) a rabies-infected area. – **Exposed:** A person who had close contact (usually a bite or a scratch) with a laboratory-confirmed rabid animal.
Mode of transmission	Usually through the bite of an infected mammalian species (dog, cat, fox, bats): bites or scratches introduce virus-laden saliva into the human body. No human-to-human transmission has been documented.
Incubation	The incubation period usually ranges from 2 to 10 days but may be longer (up to 7 years).
Period of communicability	In dogs and cats, usually for 3–7 days before onset of clinical signs (rarely more than 4 days) and throughout the course of the disease. Longer periods of excretion before onset of clinical signs have been observed in other animals.

EPIDEMIOLOGY

Burden	In 2000: • **Total number of rabies deaths in humans:** 32 (diagnosed on clinical grounds only) – Human rabies diagnosis in Sudan is usually based on clinical grounds only. Laboratory confirmation is very rare. – 8934 persons received post-exposure treatment; 100% of them received vaccine alone. • **Total number of animal rabies cases:** – 35 (confirmed by laboratory examination) – 189 (diagnosed on clinical grounds only) In 2002, 42 unprovoked bites from suspected rabid dogs and 3 deaths from rabies were confirmed in southern Sudan. In Sudan, canine rabies remains the most important epidemiological type of the disease: dogs represent more than 90% of human exposure. The spread of rabies in Sudan increased in 2000 by more than 10% compared with 1999. Rabies foci occur in most parts of the country. Dog vaccination is optional in Sudan. The total number of dogs vaccinated is 2946. Estimated dog vaccination coverage is 5%. Vaccine is also applied to cats, monkeys and bovines.
Geographical distribution	Foci of human disease occur in limited areas within the country. Foci of animal disease occur in most parts of the country.
Seasonality	No seasonality reported.
Alert threshold	One case in a susceptible animal species and/or human must lead to an alert.
Recent epidemics	**August–October 2002***:* suspected rabies outbreak in Rumbek, Twic and Ikotos/Torit (Lakes and Western Equatoria states). A total of 38 unprovoked dog bites and 3 deaths with manifestations consistent with rabies among people were reported. Suspected rabid dogs were killed but no specimens were tested. Bitten people were vaccinated.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**No**	
Overcrowding	**Yes**	An infected animal has the potential to bite more people due to an increased dog population density parallel to humans.
Poor access to health services	**Yes**	Prompt administration of vaccine post exposure (plus immunoglobulin if heavy exposure) is the only way to avoid death of an infected person.
Food shortages	**No**	
Lack of safe water and poor sanitation	**No**	
Others	**Yes**	Availability of food sources for the dogs and wild susceptible animals increases their number. Children aged 5–15 years are the group at major risk.
Risk assessment conclusions	Risk of epidemics for humans is significant if cases of rabies are reported in dogs or other susceptible animals in the same zone.	

PREVENTION AND CONTROL MEASURES

Case management

There is no specific treatment for rabies, which is a fatal disease.

The most effective way to prevent rabies after exposure is to wash and flush the wound or point of contact with soap and water, detergent or plain water, then apply ethanol or tincture or aqueous solution of iodine. Anti-rabies vaccine should be given for Category II and III exposures as soon as possible, according to WHO recognized regimens (see below). Anti-rabies immunoglobulin should be applied for Category III exposures only. Suturing should be postponed if possible; if it is necessary, immunoglobulin must be applied first. Where indicated, antitetanus treatment, antimicrobials and drugs should be administered to control infections other than rabies.

Recommended treatments according to type of contact with suspect animal

Category	Type of contact with a suspect or confirmed rabid domestic or wild animal, or animal unavailable for testing	Type of Exposure	Recommended treatment.
I	Touching or feeding of animals; Licks on intact skin.	None	None, if reliable case history is available.
II	Nibbling of uncovered skin Minor scratches or abrasions without bleeding	Minor exposure	Administer vaccine immediately Stop treatment if animal remains healthy throughout an observation period of 10 days or if animal is humanely killed and proven to be negative for rabies by a reliable laboratory using appropriate diagnostic techniques.
III	Single or multiple transdermal bites or scratches, licks on broken skin Contamination of mucous membrane with saliva (i.e. licks) Exposures to bats	Severe exposure	Administer rabies immunoglobulin and vaccine immediately. Stop treatment is animal remains healthy throughout an observation period of 10 days or if animal is humanely killed and found to be negative for rabies using appropriate diagnostic techniques

Epidemic control

Immediate notification if one or more suspected cases are identified.
Confirm the outbreak in accordance with WHO guidelines.
Confirm diagnosis and ensure prompt management.

Prevention

WHO promotes human rabies prevention through:

- Well-targeted post exposure treatment using modern vaccine types and, when appropriate, antirabies immunoglobulin
- Increased availability of modern rabies vaccine.

Elimination of dog rabies through mass vaccination of dogs and dog population management.

Immunization	Preventive mass vaccination in humans is generally not recommended but can be considered under certain circumstances for the age group 5–15 years.

21. SCHISTOSOMIASIS

DESCRIPTION

Infectious agent	Helminths: *Schistosoma haematobium* (agent of urinary schistosomiasis) and *Schistosoma mansoni* (agent of intestinal schistosomiasis), blood fluke worms belonging to the class Trematoda. Other *Schistosoma* species are not present in Sudan.
Case definition	**URINARY SCHISTOSOMIASIS** 1. ENDEMIC AREAS (MODERATE OR HIGH PREVALENCE) **Suspected:** Not applicable. **Probable:** Not applicable. **Confirmed:** A person with: – visible haematuria **or** – positive reagent strip for haematuria **or** – *S. haematobium* eggs in urine (microscopy). 2. NON-ENDEMIC AREAS AND AREAS OF LOW PREVALENCE **Suspected:** A person with: – visible haematuria **or** – positive reagent strip for haematuria, **and** – possible contact with infective water. **Probable:** Not applicable. **Confirmed:** A person with *S. haematobium* eggs in urine (microscopy). **INTESTINAL SCHISTOSOMIASIS** 1. ENDEMIC AREAS (MODERATE OR HIGH PREVALENCE) **Suspected:** A person with nonspecific abdominal symptoms, blood in stool, hepato(spleno)megaly. **Probable:** A person who meets the criteria for presumptive treatment, according to the locally applicable diagnostic algorithms. **Confirmed:** A person with *S. mansoni* eggs in stools (microscopy). 2. NON-ENDEMIC AREAS AND AREAS OF LOW PREVALENCE **Suspected:** A person with nonspecific abdominal symptoms, blood in stool, hepatosplenomegaly **and** possible contact with infective water. **Probable:** Not applicable. **Confirmed:** A person with *S. mansoni* eggs in stools (microscopy).
Mode of transmission	Water-based disease: Penetration of human skin by larval worms (cercariae) developed in snail after the eggs have been discharged in urine (S. *haematobium)* or faeces (*S. mansoni)* into a body of fresh water by patients with chronic schistosomiasis.
Incubation	Within 4 days: localized dermatitis at the site of cercarial penetration. Within 2–8 weeks: acute febrile reaction (Katayama fever; almost completely absent in *S. haematobium* infection). From 3 months to several years: manifestations of chronic illness.
Period of communicability	As long as eggs are discharged by patients; may be 10–12 weeks to more than 10 years after infection.

EPIDEMIOLOGY

Burden	***S. haematobium* prevalence of infection:** — Gezira: 0.5–30% — Khartoum: 10–24.9% — White Nile: 12–46% — Southern Sudan: a school-based survey conducted in Lui (Mundri county, Western Equatoria) in 2002 on 200 schoolchildren, found no cases of S. *haematobium* infection. Another survey conducted in Nyal (Upper Nile) found that the prevalence among 200 schoolchildren was 73%. — Other regions: no data available ***S. mansoni* prevalence of infection:** – Gezira: 2.2–75% – Khartoum: 0–39.9% – River Nile: 25–49.9% – White Nile: 4.6–24.9% Southern Sudan: – Eastern Equatoria: 0–9.9% – Bahr Al Jebel: in Juba area, a study conducted in 1998 among 2789 children found that the prevalence of *S. mansoni* was 6.9%. – Western Equatoria: a school-based survey conducted in Lui, Mundri county (2002) among 200 schoolchildren, obtained the following results: ▪ Prevalence of infection: 51.5% ▪ Patients with low intensity of infection (1–99 eggs/g): 52.5% ▪ Patients with moderate intensity of infection (100–399 eggs/g): 32% ▪ Patients with high intensity of infection (more than 399 eggs/g): ▪ 15.5% – Upper Nile: a school-based survey conducted in Nyal (2002) found that the prevalence of *S. mansoni* among 200 schoolchildren was 70%.
Geographical distribution	The endemic area of *S. haematobium* is located in the central part of Sudan, between the ninth and the sixteenth parallels. Those areas most affected include: – The southern two-thirds of Northern Darfur, except in the heart of the Jebel Marra mountains. – Southern Darfur. – Southern Kordofan. South of 9 °N latitude, foci of transmission are sporadic; foci north of 16 °N latitude are found only in the Nile valley. *S. mansoni* is markedly present in the southern edge of Sudan, and in the area between the two branches of the Nile (Gezira El Manaqil area). In the east of the country, there are a few foci of transmission in the zone of Khasm el Girba. In the west, some foci are found in the Jebel Marra mountains. Foci of transmission are also present along the White Nile. Few data are available on the epidemiology of *S. mansoni* in southern Sudan, but prevalence is known to be high. Generally speaking, the most humid and the coolest regions (River Nile tributaries and the irrigation canals appear to be more susceptible to the spread of intestinal schistosomiasis, and the arid areas to the spread of urinary schistosomiasis. The major foci of transmission are invariably in the savannah zone.
Seasonality	Dry periods tend to increase transmission of the disease as a result of higher cercarial densities in bodies of water and to drying of wells with consequent increased use of unsafe water.

Recent epidemics in the country	Schistosomiasis is usually an endemic disease, with little likelihood of rapid changes in incidence. Surveys may identify areas of particularly high endemicity where mass treatment will be warranted.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Population displacement in Somalia and Sudan is known to have led to the introduction of *S. mansoni* in areas previously endemic for *S. haematobium* only.
Overcrowding	**Yes**	Higher population densities increase the likelihood of snails being penetrated and colonized *by* miracidia.
Poor access to health services	**Yes**	Regular treatment of cases has proved effective in reducing or preventing introduction of *Schistosoma* spp. into *Schistosoma*-free areas.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	Use of surface water infested by cercariae and contamination of water by urination/defecation are essential for transmission of schistosomiasis.
Others	**No**	
Risk assessment conclusions		In chronic complex emergencies such as the one affecting Sudan, case management and control of schistosomiasis should be a priority intervention due to the effect that this disease plays on the general status of infested individuals and on the increased severity of concomitant infections. Infected individuals are predisposed to more severe diarrhoea, anaemia and abdominal symptoms. No large-scale programmes are currently implemented in Sudan. Praziquantel is locally produced and is available commercially. However, the quality of the drug is not always good, and should be tested before being used in control programmes.

PREVENTION AND CONTROL MEASURES

Case management	Praziquantel is the drug of choice against all schistosome parasites. A single oral dose of 40 mg/kg is generally sufficient to produce cure rates of 80–90% and dramatic reductions in the average number of eggs excreted. Praziquantel treatment for 1 person requires, on average, 3 tablets of 600 mg in 1 dose. The cost of a tablet is now less than US$ 0.10, bringing the total drug cost of a treatment to about US$ 0.35.

Prevention

1. Regular treatment of individuals according to the following community categories:

Community diagnosis (through primary-school surveys) and treatment regimen for schistosome infections

Community category	**Prevalence**
I – high prevalence	≥30% visible haematuria (*S. haematobium* by questionnaire) OR ≥50% infected (*S. mansoni*, *S. haematobium* by parasitological methods)
II – moderate prevalence	<30% visible haematuria (*S. haematobium*, by questionnaire) OR ≥10% but <50% infected (*S. mansoni*, *S. haematobium*, by parasitological methods)
III – low prevalence	<10% infected (*S. haematobium, S. mansoni*, by parasitological methods)

Category I:

- *Intervention in schools (enrolled and non-enrolled children):* Targeted treatment of school-age children, once a year.
- *Health services and community-based intervention:* Access to praziquantel for passive case treatment + community-directed treatment for high-risk groups* is recommended.

* Such groups include pre-school children, school-age children, pregnant women and workers with occupations involving contact with fresh water.

Category II:

- Intervention in schools (enrolled and non-enrolled children): Targeted treatment of school-age children, once every 2 years.
- *Health services and community-based intervention:* Access to praziquantel for passive case treatment.

Category III:

- *Intervention in schools (enrolled and non-enrolled children):* Targeted treatment of school-age children twice during primary schooling (once on entry, again on leaving).
- *Community-based intervention:* Access to praziquantel for passive case treatment.

For the definition of classes of intensity and further information, see *Prevention and control of schistosomiasis and soil-transmitted helminthiasis. Report of a WHO Expert Committee*. Geneva, WHO, 2002 (WHO Technical Report Series, No. 912).

2. Creation of alternative, safe water sources to reduce infective water contact.
3. Proper disposal of faeces and urine to prevent viable eggs from reaching bodies of water containing snail hosts.
4. Health education to promote early care-seeking behaviour, use of safe water (if available) and proper disposal of excreta.
5. Reduction of snail habitat and snail contact (in irrigation and agriculture practices); environmental management.
6. Treatment of snail breeding sites with molluscicides (if costs permit).

22. SOIL-TRANSMITTED HELMINTHIASES (ascariasis, hookworm infection, trichuriasis)

DESCRIPTION

Infectious agent	Helminths: *Ascaris lumbricoides*, hookworm, *Trichuris trichiura*
Case definition	• **Ascariasis:** Suspected: Abdominal or respiratory symptoms **and** history of passing worms. Confirmed: Suspected case **and** passage of *A. lumbricoides* (anus, mouth, nose), **or** presence of *A. lumbricoides* eggs in stools (microscopy). • **Hookworm infection:** Suspected: Severe anaemia for which there is no other obvious cause. Confirmed: Suspected case **and** presence of hookworm eggs in stools (microscopy). • **Trichuriasis:** Suspected: Bloody, mucoid stools. Confirmed: Suspected case **and** presence of *T. trichiura* eggs in stools.
Mode of transmission	– Ingestion of eggs, mainly as a food contaminant: *A. lumbricoides* and *T. trichiura* – Active penetration of skin by larvae in the soil: Hookworm
Incubation	– 4–8 weeks for *A. lumbricoides* – a few weeks to many months for hookworm – unspecified for *T. trichiura*.
Period of communicability	– *A. lumbricoides:* eggs appear in the faeces 45–75 days after ingestion and become infective in soil after 2–3 weeks. They can remain viable in soil for years. Infected people can contaminate soil as long as mature fertilized female worms live in the intestine (lifespan of adult worms can be 12–24 months). – Hookworm*:* eggs appear in the faeces 6–7 weeks after infection. As larvae they become infective in soil after 7–10 days and can remain infective for several weeks. Infected people can contaminate soil for several years. – *T. trichiura:* eggs appear in the faeces 70–90 days after ingestion and become infective in soil after 10–14 days. Infected people can contaminate soil for several years.

EPIDEMIOLOGY

Burden	**1992.** A survey carried out in Juba (Bahr Al Jebel) obtained 241 faecal samples and gave the following prevalence rates: *Ascaris lumbricoides*: 1.2%. *Trichuris trichiura*: 0.8%. Hookworm: 36%. **1994**. A survey conducted in 6 different localities in Sudan covering all climatic conditions gave the following results (2489 faecal samples): Geohelminths: 53 cases (2.12%). 50 out of 53 cases were from Juba (Bahr Al Jebel). Another survey conducted in Juba, Bahr Al Jebel (2789 stool samples) found a prevalence of geohelminthic infections of 20.6%. **1998.** A study performed in Chukudum and Kimatong (Eastern Equatoria) gave the following results (274 faecal samples): *Ascaris lumbricoides:* not detected *Trichuris trichiura:* 5 cases (1.8%). Hookworm: 36 cases (13.1 %).
Geographical distribution	Soil-transmitted helminthiases are mainly present in southern Sudan.
Seasonality	No data available.
Recent epidemics in the country	Soil-transmitted helminthiases are usually endemic diseases, with little likelihood of rapid changes in incidence. Surveys may identify areas of particularly high endemicity where mass treatment will be warranted.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Strictly linked to insufficient sanitation resources. Not a risk factor if people remain in the same place for a period shorter than the time needed for eggs to be discharged by an infected patient and become infective themselves (at least 45–50 days).
Overcrowding	**Yes**	Linked to the number of people defecating and to unsafe faeces disposal.
Poor access to health services	**Yes**	No treatment provided.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	The number of people relative to available sanitation facilities is the most important risk factor.
Others	**No**	

Risk assessment conclusions	Control of helminthic infestations can play a major role in reducing the communicable disease burden shouldered by populations in complex emergency situations. Moreover, given their simplicity and feasibility, intestinal helminth control activities can represent a starting point for the reconstruction of health care systems in complex emergency-affected countries. All soil-transmitted helminthiases compete with the host for nutrients, causing malabsorption of fats, proteins, carbohydrates and vitamins, and directly synergizing malnutrition. They can cause growth retardation. ***A. lumbricoides*** infestation exacerbates vitamin A deficiency. Elimination of ascarids may therefore result in rapid clinical improvement in night blindness and dryness around the eye. Infection from measles in a patient already infected with *A. lumbricoides* can result in a very severe disease. **Hookworm** infestation is strongly associated with chronic anaemia. Significant inverse correlations between intensity of worm infestation and haemoglobin level have been demonstrated. Heavy ***T. trichiura*** infection may cause diarrhoea and severe malabsorption. There is currently no national programme for the control of soil-transmitted helminthiases in Sudan. Soil-transmitted helminths can be controlled with low-cost, highly effective interventions that can dramatically increase the quality of life of affected populations. The average cost in a school distribution campaign (including drugs, distribution and monitoring activities) is approximately US$ 0.05 per child.

PREVENTION AND CONTROL MEASURES

Case management	For treatment, WHO recommends the following four drugs: **albendazole** 400 mg, **or** **levamisole** 2.5 mg/kg, **or** **mebendazole** 500 mg, **or** **pyrantel** 10 mg/kg (less commonly used because it is more difficult to administer) ***Note 1****: These drugs must not be given during the first trimester of pregnancy.* ***Note 2:*** *Where mass treatment with albendazole for filariasis is envisaged, chemotherapy of intestinal helminths will take place as part of the antifilarial chemoprophylaxis.*
Prevention and control	**Overall:** Personal hygiene, disposal of faeces, hand-washing and clean food Improvements in sanitation standards (see Appendix 3: *Safe water and sanitation*) Community-wide treatment according to the following categories: Community diagnosis (through primary-school surveys) and treatment regimen for STH:

Community category of any infection	**Prevalence**	**% of moderate-to-heavy intensity infections**
I (high prevalence–high intensity)	≥70%	≥10%
II (high prevalence–-low intensity)	≥50% but <70%	<10%
III (low prevalence–low intensity)	<50%	<10%

	Category I: • *Intervention in schools (enrolled and non-enrolled children):* Targeted treatment of school-age children, 2–3 times a year. • *Health services and community-based intervention:* Systematic treatment of pre-school children and women of childbearing age in mother and child health programmes. **Category II:** • *Intervention in schools* (enrolled *and non-enrolled children):* Targeted treatment of school-age children, once a year. • *Health services and community-based intervention:* Systematic treatment of pre-school children and women of childbearing age in mother and child health programmes. **Category III:** • *Intervention in schools (enrolled and non-enrolled children):* Selective treatment. • *Community-based intervention:* Selective treatment. For the definition of classes of intensity and further information, see: *Prevention and control of schistosomiasis and soil-transmitted helminthiasis. Report of a WHO Expert Committee*. Geneva, WHO, 2002 (WHO Technical Report Series, No. 912). **In case of suspected or confirmed hookworm infection, in addition:** • In highly endemic areas, wear shoes. • Consider drug treatment and iron supplementation during pregnancy.

23. TUBERCULOSIS

DESCRIPTION

Infectious agent	Bacterium: *Mycobacterium tuberculosis.* This complex includes *M. tuberculosis* and *M. africanum* primarily from humans, and *M. bovis* primarily from cattle.
Diagnosis in Adults	**Clinical description** The most important symptoms in the selection of tuberculosis (TB) suspects in adults (aged older than 15 years) are: – productive cough for more than 2 weeks, **and/or** – haemoptysis **and** – significant weight loss. Patients with TB may also have other symptoms (which are more common, but less suggestive) such as: – chest pain – breathlessness – fever/night sweats – tiredness, **and** – loss of appetite. **Among refugee and internally displaced populations**, it is unusual to have ready access to X-ray facilities. It is the priority of health services to detect the sources of infection by sputum microscopy, and cure them. **Clinical case definition** **Tuberculosis suspect:** Any person who presents with symptoms or signs suggestive of TB, in particular cough of long duration (more than 3 weeks) **Case of tuberculosis:** A patient in whom TB has been bacteriologically confirmed or diagnosed by a clinician. ***Note:*** *Any person given treatment for TB should be recorded as a case. Incomplete "trial" tuberculosis treatment should not be given as a method for diagnosis.* **Definite case of tuberculosis:** A patient with positive culture for the *M. tuberculosis* complex. (In countries where culture is not routinely available, a patient with two sputum smears positive for acid-fast bacilli (AFB) is also considered a "definite" case.) **Laboratory criteria for diagnosis** Each TB suspect should have three sputum samples examined by light binocular microscopy for AFB. The chances of finding TB organisms are greater with three sputum samples than with one or two samples. Secretions build up in the airways overnight, so that an early-morning sputum sample is more likely to contain the TB organism than a sample taken later in the day. In practice, a suspect provides sputum samples in the following manner:

	Day 1 **Sample 1** – Person suspected of TB provides an "on-the-spot" sample under supervision on presentation to the health facility. He or she is given a sputum container to take home for an early-morning sample the following day. **Day 2** **Sample 2** – Person suspected of TB brings an early-morning sputum sample collected just after waking up. **Sample 3** – Person suspected of TB provides another "on-the-spot" sample. **At least two sputum smears are positive** Smears should be stained using the Ziehl–Neelsen method. Any TB suspect with two positive smears is a smear-positive TB patient, who must then be registered and started on anti-TB treatment. **If only one initial sputum smear is positive** A suggestive X-ray showing active pulmonary TB interpreted by an experienced medical officer may lead to a diagnosis of smear-positive TB. AFB microscopy may be repeated and, if at least one smear is again positive with compatible X-ray, the patient should be considered a smear-positive TB patient. In the absence of X-ray, one sputum smear with positive culture for *M. tuberculosis* is also classified as sputum-positive TB. **If all three sputum smears are negative** If the initial three smears are negative, but pulmonary TB is still suspected because of persistent symptoms, the suspect should be treated for acute respiratory infection with broad-spectrum antibiotics (e.g. amoxicillin or co-trimoxazole, **but not rifampicin or any other anti-TB drug**) for at least 1 week. If there is no improvement, sputum samples must be re-examined 2 weeks after the first sputum examination. Between 65–80% of all pulmonary TB cases are expected to be confirmed by positive sputum smear examination. X-ray lesions compatible with active TB should encourage further sputum examination if the three sputum smear examinations were negative. X-ray itself is not a diagnostic tool for pulmonary TB. In *some* circumstances, a compatible X-ray together with symptoms consistent with TB will lead to the diagnosis of pulmonary TB in smear-negative cases. Thus, if all three samples are again negative after the trial of antibiotics, either a compatible X-ray interpreted by an experienced physician or, in the absence of X-ray facilities, the experienced physician's judgement alone will decide whether a patient is categorized as having TB (classed as smear-negative TB). Additional cases of TB may be found among close contacts of known smear-positive cases, either family members or persons sleeping in the same shelter. Symptomatic contacts should be screened using the procedures described above.
	<u>TB in HIV-positive patients</u> HIV-positive patients are more susceptible to TB infection, and HIV in a TB patient is a potent cause of progression of TB infection to disease. The principles of TB control are the same even when there are many HIV/TB patients. In HIV-infected patients, pulmonary TB is still the commonest form of TB. The clinical presentation of TB depends on the degree of immunosuppression. Early in HIV infection, when immunity is good, the signs of TB are similar to those in an individual without HIV infection. As HIV infection progresses and immunity declines, the risk of TB dissemination increases. TB meningitis, miliary TB and widespread TB lymphadenopathy occur. It is important to look systematically for signs or symptoms of TB in HIV-positive patients and to start treatment without delay based on clinical, bacteriological and, in some circumstances, radiological evidence.

Diagnosis in Children	TB in children is a general disease, which may affect any part of the body. Children rarely have smear-positive TB, so they are rarely infectious. In complex emergency situations with a large number of children, extrapulmonary forms of TB should be suspected, diagnosed and treated appropriately. This may often require referral to a hospital for X-ray and special examinations (e.g. lumbar puncture). In children with headache, change of temperament, recent squint or ocular muscle paralysis, or dyspnoea, meningitis should be suspected. TB is one cause of meningitis, although rare – meningococcal meningitis is more common in complex emergency settings. Definitive diagnosis requires hospital referral. Children with high fevers, dyspnoea, gastrointestinal symptoms, confusion (i.e. those with suspicion of acute miliary TB) must also be referred to hospital for assessment and diagnosis. Suspected bone and joint TB, or pleural effusions, also require referral. Commoner forms of extrapulmonary disease (e.g. cervical or auxiliary lymphadenitis, peritonitis with ascites) can be diagnosed and treated in a camp situation. The diagnosis of TB in children should be carefully considered in a child if there is: – illness lasting for more than 10 days – history of close contact with a TB patient – poor response to antibiotic therapy – poor response to 1 month of nutritional rehabilitation – weight loss or abnormally slow growth – loss of energy, or – increasing irritability and drowsiness over a period of 2 weeks. Nutritional support and rehabilitation should be given for at least 1 month to a child in whom TB is suspected. ***Note:*** *The considerations explained above for the diagnosis of TB in HIV-positive adults also apply in to children.*

Diagnostic criteria for classification of TB	**Pulmonary tuberculosis (PTB)** Pulmonary TB refers to disease involving the lung parenchyma. Tuberculous intrathoracic lymphadenopathy (mediastinal and/or hilar) or tuberculous pleural effusion, without radiographic abnormalities in the lungs, therefore constitutes a case of extrapulmonary TB. A patient with both pulmonary and extrapulmonary TB should be classified as a case of pulmonary TB. • **Smear-positive pulmonary TB** **Either:** A patient with at least two sputum specimens positive for AFB by microscopy; **or:** A patient with at least one sputum specimen positive for AFB by microscopy and radiographic abnormalities consistent with pulmonary TB; **or:** A patient with at least one sputum specimen positive for AFB by microscopy, which is culture-positive for *M. tuberculosis*. • **Smear-negative pulmonary TB** A case of PTB that does not meet the above definition for smear-positive TB. This group includes cases without smear result. This commonly occurs in children but is comparatively uncommon in adults. **Diagnostic criteria for PTB (which is also used to exclude sputum negative PTB) is based on the following criteria:** – at least three sputum specimens negative for AFB, **and** – no clinical response to a one-week course of broad-spectrum antibiotics, **and** – radiographic abnormalities consistent with active PTB, **and** – decision by a clinician to treat with a full course of anti-TB chemotherapy. A patient whose initial sputum smears were negative and whose subsequent sputum culture result is positive is also considered to have smear-negative pulmonary TB. **Extrapulmonary tuberculosis (EPTB)** EPTB refers to TB of organs other than the lungs, e.g. pleura, lymph nodes, abdomen, genitourinary tract, skin, joints and bones, meninges. Diagnosis should be based on one culture-positive specimen, or on histological or strong clinical evidence consistent with active EPTB, followed by a decision by a clinician to treat with a full course of anti-TB chemotherapy. **The case definition of an EPTB case with several sites affected depends on the site representing the most severe form of disease.** Some cases will be easy to diagnose with peripheral lymphadenitis, swelling of cervical or axillary lymph nodes, chronic evolution and/or production of caseous discharge. Other cases, such as severe, life-threatening forms (e.g. miliary TB, TB meningitis), TB of bone joints, TB peritonitis, TB laryngitis, will be suspected but should be referred to a hospital for assessment.
Mode of transmission	Exposure to tubercle bacilli in airborne droplet nuclei produced by people with pulmonary or laryngeal TB during expiratory efforts such as coughing and sneezing. Extrapulmonary tuberculosis (other than laryngeal) is usually non-infectious. Bovine tuberculosis results from exposure to tuberculous cattle, usually by ingestion of unpasteurized milk or dairy products, and sometimes by airborne spread to farmers and animal handlers.

Progression to active disease	Progression to active disease can take weeks or years; latent infections may persist throughout life. The risk of TB occurrence is relatively high during the first year following TB infection, then progressively decreases by half within the next 4–5 years. Only 10% of infected people with normal immune systems will develop clinically evident TB at some point in life; 5% will have an early progression of the disease (primary tuberculosis); the remaining 5% will have a late progression of the disease (post-primary tuberculosis) after a period of initial containment.
Period of communicability	As long as viable tuberculosis bacilli are being discharged in the sputum. Effective treatment usually eliminates communicability within 2 weeks.

EPIDEMIOLOGY

Burden	**Estimated number of new cases of all forms of TB:** **2002:** 71 211 (of which 24 554 (34.4%) were notified) **2001:** 59 897 (of which 23 997 (40.0%) were notified) **2000:** 59 875 (of which 24 807 (41.4%) were notified) **Estimated number of new cases of smear positive** (ss+) **TB:** **2002:** 31 432 (of which 33.01%) were notified) **2001:** 26 953 (of which 11 136 (41.3%) were notified) **2000:** 26 944 (of which 12 311 (45.6%) were notified) (Data source: WHO/Sudan)
Geographical distribution	Tuberculosis is known to be widespread throughout the country.
Seasonality	No specific seasonality is reported.
Alert threshold	Not applicable.
Recent epidemics in the country	Not applicable.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	May generate conditions that disrupt TB treatment.
Overcrowding	**Yes**	Overcrowding is recognized as one of the most important factors leading to increased risk of transmission.
Poor access to health services	**Yes**	– People affected by TB who cannot access health services and be treated remain infectious for a longer period. – The case-fatality rate is high (about 50%) without proper treatment. – Together with poor drug prescribing practices, the interruption of treatment is one of the most important causes of development of multidrug-resistant TB (MDR-TB).
Food shortages	**Yes**	Poor nutritional status increases vulnerability to TB infection and development of active disease.
Lack of safe water and poor sanitation	**No**	

Others	**Yes**	Low BCG coveragerates (<80%) can lead to high TB rates in children. **BCG vaccination coverage** **2001:** 71% (51% by WHO–UNICEF estimates) **2000:** 66% **1999:** 92% **1998:** 80% **1997**: 80% **1990:** 73% **1980:** 2% (Data source: WHO/Sudan country estimates)
Risk assessment conclusions	Sudan has successfully implemented TB control programmes in accordance with the DOTS strategy. However, the southern part of Sudan has not yet fully provided DOTS services. The smear-positive TB case-detection rate (new ss+ notified/new ss+ estimated) fell from 41% in 2001 to 33% in 2002. The treatment success rate was 79% in 2000 and 80% in 2001. The global target is to detect 70% of all cases and successfully treat 85% of them by 2005.	

PREVENTION AND CONTROL MEASURES

Case management	**Standardized short-course chemotherapy using regimens of 6–8 months.** Good case management includes directly-observed therapy (DOT) during the intensive phase for all new sputum-smear positive cases, the continuation phase of rifampicin-containing regimens and the whole of the re-treatment regimen. There are three main types of treatment regimens. These treatment regimen is based according to patient categories I, II and III as described below. The chemotherapeutic regimens are based on standardized combinations of 5 essential drugs: rifampicin (R), isoniazid (H), pyrazinamide (P), ethambutol (E) and streptomycin (S). Each of the standardized chemotherapeutic regimens consists of two phases: – **Initial (intensive) phase:** 2–3 months, with 3–5 drugs given daily under direct observation. – **Continuation phase:** 4–6 months, with 2–3 drugs given 3 times weekly under direct observation or, in some cases (e.g. during repatriation of displaced populations), 2 drugs for 6 months given daily, unsupervised, but in fixed-dose combinations. Staff should observe all doses of rifampicin-containing regimens; actual swallowing of medication should be checked. Hospitalized patients should be kept in a separate ward for the first 2 weeks of treatment. **Previously treated case** A patient who has at any time received anti-TB treatment for more than 1 month. This group of patients comprises: – **Return after interruption:** common among recent refugees or IDPs. – **Failure:** a patient who, while on treatment, remained, or became again, smear-positive, 5 months or later after starting treatment; also, a patient who was smear-negative before starting treatment and who became smear-positive after the second month of treatment. – **Relapse:** a patient who has been declared cured of TB in the past by a physician after a full course of chemotherapy and who has become sputum smear-positive. – **Chronic:** a patient who remained, or became again, smear-positive at the end of a fully supervised, standardized re-treatment regimen (very small number of previously treated cases).

Treatment in Children	The drug regimens used for children are the same as for adults* **Drug dosages must be calculated according to the child's weight. Adjustments may have to be made during the course of the treatment as the child may rapidly regain lost weight.** For infants of newly diagnosed smear-positive mothers, breastfeeding should continue. The infant should not be separated from the mother. Transmission is likely to have already occurred, and the infant is at greater risk of dying from other causes if breastfeeding is stopped. If the infant is well, isoniazid prophylaxis should be given for 6 months and requires regular follow-up, for example one in every two months. **See**: *Precautions for use of streptomycin and ethambutol in children.* Geneva, WHO, 2003 (WHO/CDS/TB 2003.313; page 64).
Treatment categories	Treatment categories are essential for prioritization of TB treatment according to public health risk – Category I is the highest priority. **Category I** These patients are: – smear-positive persons who have never previously been treated or who have only received treatment for less than 1 month. – severely ill patients with other forms of TB (new smear-negative pulmonary TB. with extensive parenchymal involvement, and new cases of severe forms of TB[1]). The recommended regimen lasts 6 months. The initial (intensive) phase of treatment lasts for 2 months; rifampicin, isoniazid, pyrazinamide and ethambutol are given daily or 3 times weekly (streptomycin may be used as a subsititute for ethambutol), under direct supervision. At the end of the second month of treament, most patients will have a negative result on sputum microscopy; they can then progress to the second stage of treatment – the continuation phase. This phase lasts for 4 months, with rifampicin and isoniazid given 3 times weekly, under direct supervision.[2] If the sputum smear examination is positive at the end of the second month, for whatever reason, the initial phase is prolonged for a third month. The patient then starts the continuation phase irrespective of the results of the sputum examination at the end of the third month. If the sputum smears are still positive at the end of the fifth month or at the end of a treatment regimen, the patient is classified a **treatment failure case.** He or she is re-registered and starts a full course of the re-treatment regimen as a Category II patient. Drug dose is adjusted for weight gain at the end of the initial phase (2nd or 3rd month). [1]This category includes patients with TB meningitis, disseminated TB, pericarditis, peritonitis, bilateral or extensive pleurisy, vertebral disease with neurological complications, and intestinal and genitourinary disease. [2]Daily self-administered ethambutol and isoniazid may be used in the continuation phase for 6 months, so this treatment regimen lasts a total of 8 months. However, this regimen is associated with a higher rate of failure and relapse.

Category II

Patients who were previously treated and are now sputum smear-positive include:

– treatment after interruption;
– treatment failure; and
– relapse after treatment.

These patients should receive a standardized re-treatment regimen, fully supervised throughout both phases of treatment.

The initial phase of treatment lasts for 3 months; rifampicin, isoniazid, pyrazinamide and ethambutol are given daily and supplemented by streptomycin daily for the first 2 months.

The continuation phase of this regimen constitutes 5 months of rifampicin, isoniazid and ethambutol given 3 times weekly.

Sputum smear examination is performed at the end of the initial phase of treatment (i.e. at the end of 3 months), during the continuation phase of treatment (at the end of the fifth month) and at the end of treatment (at the end of the eighth month). If the patient is sputum smear-positive at the end of the third month, the initial phase of treatment is extended with rifampicin, isoniazid, pyrazinamide and ethambutol for one more month. Patients who are still positive at the end of the fourth month progress to the continuation phase, regardless of the results of the sputum examination.

Category III

These patients include:
– smear-negative pulmonary patients (with limited parenchymal involvement)
– adults and children with non-serious extrapulmonary disease (including symptomatic primary disease).

All Category III patients should receive 2 months of rifampicin, isoniazid and pyrazinamide daily, followed by 4 months of isoniazid and rifampicin every second day.

When the continuation phase cannot be carried out under direct observation, all patients should be given daily ethambutol and isoniazid in the continuation phase for 6 months.

HIV-positive patients

Anti-TB drug treatment is the same for HIV-positive and HIV-negative patients, with one exception: thiacetazone should not be given to HIV-positive TB patients as there is increased risk of severe toxicity and sometimes fatal skin reactions.

Controlled clinical trial studies have shown that isoniazid preventive treatment (IPT) reduces the risk of TB disease in HIV-positive individuals with latent TB infection (shown by a positive tuberculin skin test).

The use of IPT has shown to be more effective than other regimens for prevention of latent TB infection. The decision to use IPT must be carefully evaluated, and requires first the exclusion of active TB in the patient.

To manage the problem of HIV/TB coinfection effectively, TB and HIV programmes should coordinate activities through a TB/HIV coordinating body.

See:

– *An expanded DOTS framework for effective tuberculosis control*. Geneva, WHO, 2002 (WHO/CDS/TB/2002.297).
– *Treatment of tuberculosis: guidelines for national programmes*, 3rd ed. Geneva, WHO, 2003 (WHO/TB/2003.313).
– *Tuberculosis control in refugee situations: an inter-agency field manual.* Geneva, WHO, 1997 (WHO/TB/97.221; to be updated in 2004).

Prevention and control	Detection and treatment of smear-positive (infectious) TB cases is the most effective preventive measure. To ensure the appropriate treatment and cure of TB patients, strict implementation of the **DOTS strategy** is important. DOTS is the internationally recommended strategy for TB control, and has the following components: – Government commitment to ensuring sustained, comprehensive TB control activities. – Case detection by sputum smear microscopy among symptomatic patients self-reporting to health services. – Standardized short-course chemotherapy using regimens of 6–8 months, with direct observation of treatment at least during the intensive phase (or for as long as rifampicin is administered) for at least all confirmed smear-positive cases (see *Case management*). – A regular, uninterrupted supply of all essential anti-TB drugs. – A standardized recording and reporting system that allows assessment of follow-up and treatment results for each patient and of the TB control programme's overall performance. **Complementary control strategies:** – Health education to improve awareness and reduce stigma. – Maintaining good ventilation and reducing overcrowding in health clinics, and ensuring hospitalized patients are kept in a separate ward for the first 2 weeks of treatment. – Isoniazid prophylaxis is not recommended in refugee situations, except for children being breastfed by smear-positive mothers. If the child is well, BCG vaccination should be postponed and isoniazid given to the child for 6 months. In the event of a sudden disruption to the programme, isoniazid may be stopped and BCG given before the child leaves the refugee camp (preferably after a one-week interval).
Immunization	BCG has been shown to be effective in preventing severe forms of TB such as TB meningitis and miliary TB in children. As overcrowding and malnutrition are common among many refugee and displaced populations, the risk of TB transmission to children is increased. BCG is strongly recommended for all newborn children and any children aged up to 5 years who have not already received it. The vaccination of newborns should be incorporated into routine immunization programmes for all children. Re-vaccination is not recommended.

Health education	Key elements of community education: — avoiding stigmatization of TB patients. — curability of TB disease. — early (self) referral of TB suspects. — importance of adherence to treatment. — contact tracing. The most important messages to teach: • TB in an adult should be suspected when the person has a productive cough lasting more than 2 weeks, and/or blood in the sputum, with significant weight loss. • Cover the mouth whenever coughing or sneezing to prevent the spread of lung diseases. • Anyone may contract TB. • TB is curable. • Early treatment is important for best results and to prevent spread, especially to family members. • Children are especially at risk if not treated and may develop severe, even fatal, disease. • Good treatment is the best prevention. • All patients must take the full course of treatment. • Treatment makes patients non-infectious in 2 weeks, but cure takes 6–8 months. • Treatment must be completed even though the patient may feel better sooner. • Failure to complete the treatment may result in a recurrence that may be impossible to treat and may spread serious disease to others, especially children. • All patients should be treated sympathetically and with respect. • Controlling TB is a community responsibility. ***Note**: Diagrams should be used as much as possible – a high literacy level should not be assumed. Cured patients are often helpful teachers and supporters of new patients.*

24. TYPHOID FEVER

DESCRIPTION

Infectious agent	Bacterium: *Salmonella eneterica* serovar Typhi *(S.*Typhi*).*
Case definition	**Clinical case definition** Clinical diagnosis is difficult. In the absence of laboratory confirmation, any case with fever of at least 38 °C for 3 or more days is considered suspect if the epidemiological context is conducive. **Confirmed case** Isolation of *S. typhi* from blood or stool cultures.
Mode of transmission	Faecal–oral route, particularly through contaminated water and food.
Incubation	Incubation period is usually 8–14 days but may be from 3 days up to 1 month.
Period of communicability	From the symptomatic period for 2 weeks; 2–5% of infected cases remain carriers for several months. Chronic carriers contribute significantly to spread of the disease.

EPIDEMIOLOGY

Burden	No data available.
Geographical distribution	No data available.
Seasonality	No data available.
Alert threshold	Two or more linked cases.
Recent epidemics in the country	No data available.

RISK FACTORS FOR INCREASED BURDEN

Population movement	**Yes**	Dissemination of multidrug-resistant strains of *S. typhi*.
Overcrowding	**Yes**	Very important.
Poor access to health services	**Yes**	Early detection and containment of cases are paramount in reducing dissemination. The case-fatality rate is high (10–20%) without proper treatment.
Food shortages	**No**	
Lack of safe water and poor sanitation	**Yes**	The most important risk factor.
Others	**Yes**	Multidrug-resistant strains of *S. typhi*, including resistance to ciprofloxacin. Milk and dairy products are an important source of infection.

Risk assessment conclusions	Among the general population the risk is related to the availability of safe food, clean water and sanitation. This must be ensured to prevent outbreaks. It is essential that health care workers are trained and have a reasonable degree of suspicion. Monitoring of antibiotic resistance is essential. Early and effective treatment is crucial for maintaining low case-fatality rates.

PREVENTION AND CONTROL MEASURES

Case management	Early antimicrobial treatment, selected according to the antimicrobial resistance pattern of the strain. Quinolones (e.g. ciprofloxacin), co-trimoxazole, chloramphenicol and ampicillin are usually used for typhoid fever. Dehydration prevention and case management using ORS also play an important role.
Epidemic control	Inform the health authorities when one or more suspected cases are identified. Confirm the outbreak in accordance with WHO guidelines. Confirm the diagnosis and ensure prompt treatment.
Prevention	Good sanitation can markedly reduce the risk of transmission, especially where its absence may lead to contamination of clean water sources. High priority should be given to observing the basic principles of sanitary human waste disposal as well as to ensuring the availability of safe water supplies. Appropriate facilities for human waste disposal are a basic need of all communities. The absence of such facilities creates a high risk for disease transmission. Sanitary systems that are appropriate for local conditions should be constructed with the cooperation of the community. People will need to be taught how to use latrines, about the dangers of defecating on the ground, or in or near waters, and about the importance of thorough hand-washing with soap or ash after any contact with excreta. The disposal of children's excreta in latrines needs to be emphasized.
Immunization	Mass immunization may be an adjunct for the control of typhoid fever during a sustained, high-incidence epidemic. This is especially true when access to well functioning medical services is not possible or in the case of a multidrug-resistant strain. A parenteral vaccine containing the polysaccharide Vi antigen is the vaccine of choice among displaced populations. An oral, live vaccine using *S. typhi* strain Ty21a is also available. Neither the polysaccharide vaccine nor the Ty21a vaccine is licensed for children aged under 2 years. The Ty21a vaccine should not be used in patients receiving antibiotics.

25. YELLOW FEVER

DESCRIPTION

Infectious agent	Yellow fever virus, belonging to the Flavivirus group.
Case definition	**Clinical description:** Characterized by acute onset of fever followed by jaundice within 2 weeks of onset of first symptoms. Haemorrhagic manifestations and signs of renal failure may occur. There are two disease phases for yellow fever: **Acute phase**: While some infections have no symptoms whatsoever, this first phase is normally characterized by fever, muscle pain (with prominent backache), headache, shivers, loss of appetite, nausea and/or vomiting. Often, the high fever is paradoxically associated with a slow pulse (Faget's sign). Most patients improve after 3–4 days and their symptoms disappear, but 15% enter the toxic phase. **Toxic phase**: Fever reappears; the patient rapidly develops jaundice and complains of abdominal pain with vomiting. Bleeding can occur from mouth, nose, eyes and/or stomach. Once this happens, blood appears in the vomit and faeces. Kidney function deteriorates; this can range from abnormal protein levels in the urine (albuminuria) to complete renal failure with no urine production (anuria). Half the patients in the toxic phase die within 10–14 days. The remainder recovers without significant organ damage. **Laboratory criteria:** Isolation of yellow fever virus, **or** Presence of yellow-fever-specific IgM or a fourfold or greater rise in serum IgG levels in paired sera (acute and convalescent), **or** Positive postmortem liver histopathology, **or** Detection of yellow fever antigen in tissues by immunohistochemistry, **or** Detection of yellow fever virus genomic sequences in blood or organs by polymerase chain reaction. **Case classification:** **Suspected:** a case that is compatible with the clinical description. **Probable:** not applicable **Confirmed:** a suspected case that is laboratory-confirmed (national reference laboratory) or epidemiologically linked to a confirmed case or outbreak.
Mode of transmission	Bite of infective mosquitoes. The vectors of yellow fever in forest areas in Africa are *Aedes africanus* and other *Aedes* species. In urban areas, the vector is *Ae. aegypti* (all-day biting species).
Incubation	From 3 to 6 days.
Period of communicability	Blood of patients is infective for mosquitoes shortly before onset of fever and for the first 3–5 days of illness. The disease is highly communicable where many susceptible people and abundant vector mosquitoes coexist; it is not communicable by contact or other common means of disease transmission. Once infected, mosquitoes remain so for life.

EPIDEMIOLOGY

Burden	**2001:** 0 cases reported **2000:** no data available **1999:** no data available **1998:** no data available (Source: WHO/IVB data, 2004) **1997**: no data available **1990:** no data available **1980:** no data available
Geographical distribution	The southern half of the country was officially endemic in the year 2000.
Seasonality	In forest areas, where the yellow fever virus circulates between mosquitoes and monkeys or chimpanzees, the disease is present throughout the year. In field or savannah areas outside the forest areas, the virus remains dormant in infected mosquito eggs throughout the dry season and emerges in the rainy season when eggs hatch.
Alert threshold	One confirmed case must lead to alert. An outbreak of yellow fever is at least one confirmed case.
Recent epidemics in the country	Sudan is known to be endemic. **2003**: From 11 to 15 May, 178 cases and 27 deaths occurred in Imatong and Ikotos districts, Torit county, in the south-eastern part of the country. International technical assistance, vaccine and vaccine supplies for a vaccination campaign targeting 100 000 people in the area were provided. No details of previous epidemics are available.

RISK FACTORS FOR INCREASED BURDEN

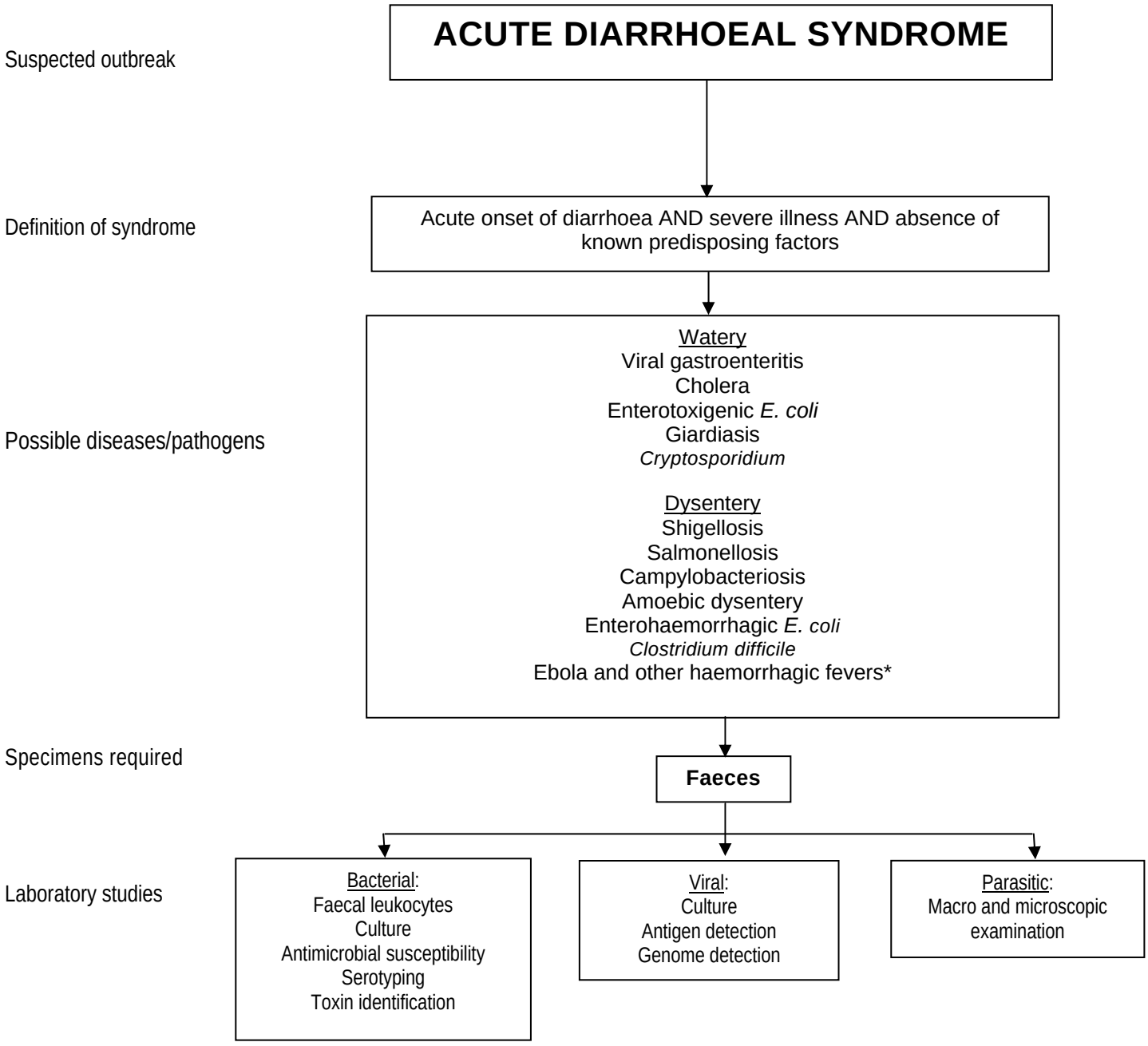
Population movement	**Yes**	Unvaccinated people moving to areas of endemicity are at risk. Changes in land use are a risk factor.
Overcrowding	**Yes**	Increased population density and increased exposure to mosquito bites in temporary shelters.
Poor access to health services	**Yes**	Collapse of vaccination programmes. Increased fatality due to unavailability of case management.
Food shortages	**No**	
Lack of safe water and poor sanitation	**No**	
Others	**Yes**	Open water storage provides a favourable habitat for *Ae. Aegypti*. Old tyres, old water containers, etc. increase vector breeding sites. Temporary surface-water bodies (poor drainage leading to pools and open channels of water) may increase vector breeding opportunities. **Yellow fever virus vaccine (YFV) coverage:** **2001:** no data available **2000:** no data available **1999:** no data available **1998:** no data available **1997:** no data available **1990:** no data available **1980:** no data available (Data source: WHO/IVB data)

Risk assessment conclusions	Although no official cases are reported from Sudan, the risk of transmission exists. Reduced coverage rates for yellow fever immunizations and disruption of mosquito control programmes are likely to have increased this risk. Moreover, population movements from rural to urban areas have resulted in large numbers of people living in conditions of poverty, overcrowding and poor sanitation, all conditions that amplify the risk of transmission. The precise extent of illness and death due to yellow fever is unknown. Disease surveillance is not adequate to detect cases of sylvatic yellow fever that often occur in remote areas. Moreover, an outbreak of yellow fever can go undetected because the signs and symptoms of yellow fever have a wide spectrum and overlap with those of many other diseases, making it difficult for health workers to make a definitive diagnosis on this basis alone. Mild cases can go undetected because patients are likely to be treated at home and may not seek care in a health facility.

PREVENTION AND CONTROL MEASURES

Case management	No specific treatment for yellow fever is available. Dehydration and fever can be corrected with oral rehydration salts. Intensive supportive care may improve the outcome but is rarely available. See: *Case management of epidemic-prone diseases* in this Toolkit for Sudan (Document 6).
Epidemic control	An infected mosquito spreads yellow fever when it bites non-infected humans. When human-to-human transmission is established, the conditions for an epidemic are in place. Depending on the travel patterns of infected humans or infected mosquitoes, the epidemic spreads from village to village and into cities. Under epidemic conditions, the following must be implemented: – Mass vaccination with YFV – Emergency mosquito control measures: • Eliminating potential mosquito breeding sites (the most important measure) • Spraying to kill adult mosquitoes (less important because of limited impact) • Use of insecticide-treated nets.
Prevention	**Vaccination is the single most important measure for preventing yellow fever.** In endemic areas, vaccination must be given routinely through the incorporation of YFV in routine child immunization programmes and mass preventive campaigns. YFV is not recommended for symptomatic HIV-infected persons or other immunosuppressed individuals. For theoretical reasons, it is not recommended for pregnant women. Recommended strategies: –vaccinating the population aged older than 9 months in counties where coverage in recent campaigns achieved less than 80%; –if funds are limited, a cheaper intervention would be to vaccinate children aged between 9 months and 14 years to reach at least 50% of the population; –yellow fever vaccination should be integrated in routine EPI activities. **Routine mosquito control measures** Potential mosquito breeding sites must be eliminated.

APPENDIX 1: FLOWCHARTS FOR THE DIAGNOSIS OF COMMUNICABLE DISEASES



*Ebola and other haemorrhagic fevers may initially present as bloody diarrhoea. If such an etiology is suspected, refer to "Acute haemorrhagic fever syndrome" for appropriate specimen collection guideline.

Suspected outbreak

ACUTE HAEMORRHAGIC FEVER SYNDROME

Definition of syndrome

Acute onset of fever of less than 3 weeks' duration AND any two of the following:

- Haemorrahgic or purpuric rash
- Epistaxis
- Haemoptysis
- Blood in stool
- Other haemorrhagic symptom

AND absence of known predisposing factors

Possible diseases/pathogens

Dengue haemorrhagic fever and shock syndrome
Yellow fever
Other arboviral haemorrhagic fevers (e.g. Rift Valley, Crimean–Congo, Tick-borne flaviviruses)
Lassa fever and other arenoviral haemorrhagic fevers
Ebola or Marburg haemorrhagic fevers
Haemorrhagic syndrome (hantaviruses)
Malaria
Relapsing fever

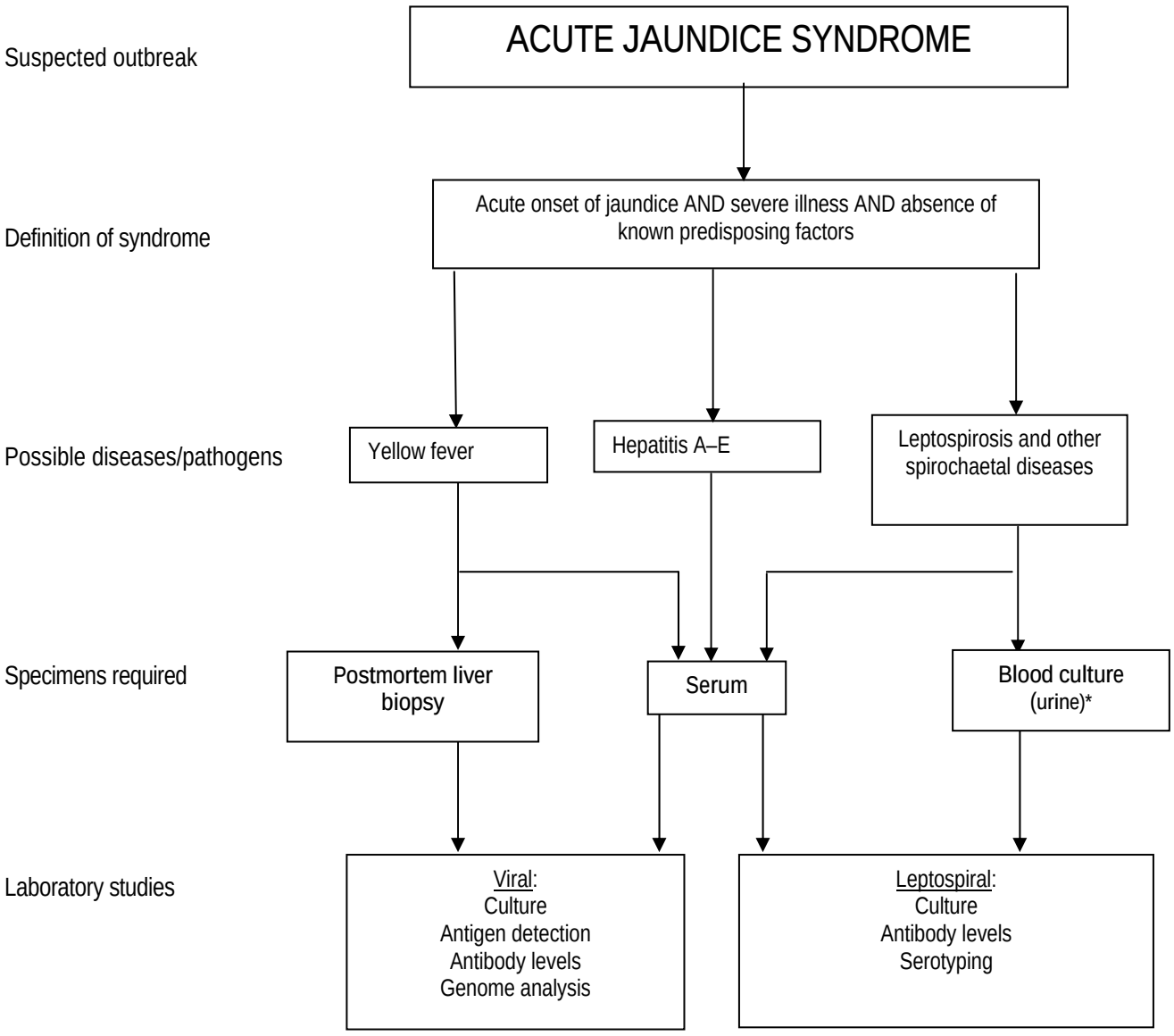
Specimens required

Blood
Blood smear
Serum
Postmortem tissue specimens (e.g. skin biopsy and/or liver biopsy)

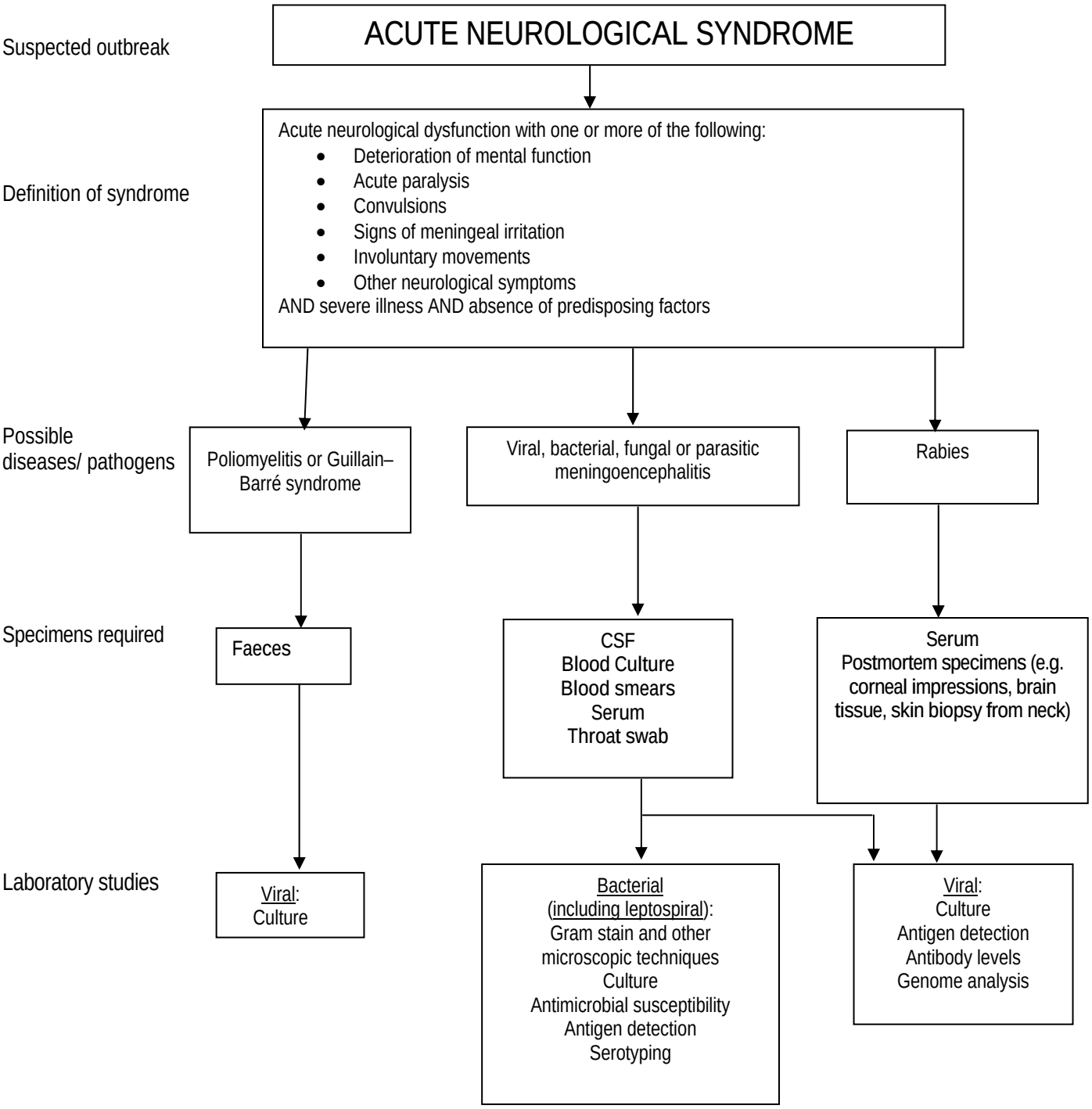
Laboratory studies

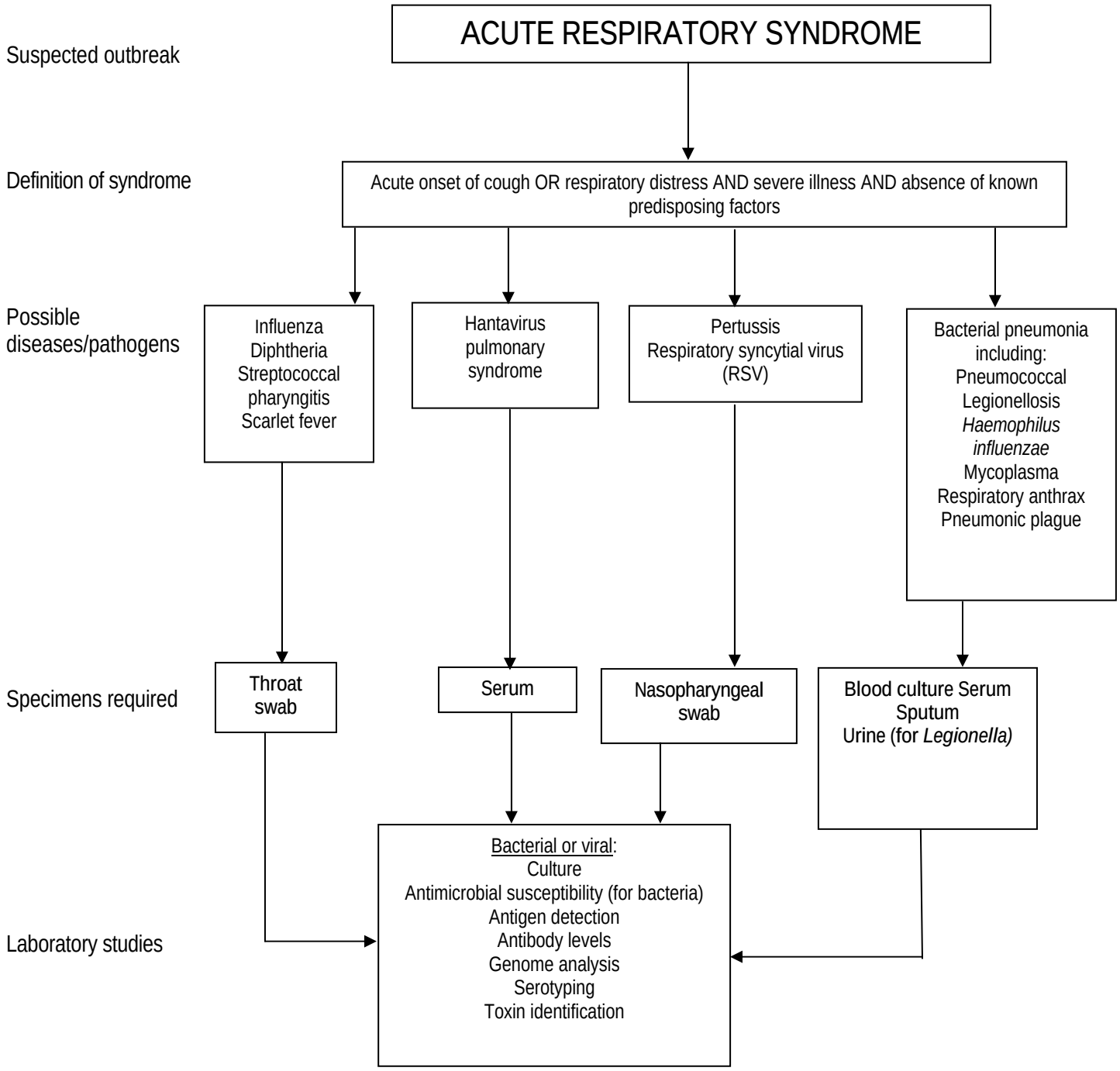
<u>Viral</u>:
Culture
Antigen detection
Antibody levels
Genome detection

<u>Parasitic</u>:
Demonstration of pathogen



** Requires specialized media and handling procedures. See Annex 7 "Guidelines for collections of speciments for laboratory testing in this Toolkit..*


Suspected outbreak
ACUTE NEUROLOGICAL SYNDROME
Definition of syndrome
Acute neurological dysfunction with one or more of the following:
Deterioration of mental function
Acute paralysis
Convulsions
Signs of meningeal irritation
Involuntary movements
Other neurological symptoms
AND severe illness AND absence of predisposing factors
Possible diseases/ pathogens
Poliomyelitis or Guillain–Barré syndrome
Viral, bacterial, fungal or parasitic meningoencephalitis
Rabies
Specimens required
Faeces
CSF
Blood Culture
Blood smears
Serum
Throat swab
Serum
Postmortem specimens (e.g. corneal impressions, brain tissue, skin biopsy from neck)
Laboratory studies
Viral:
Culture
Bacterial
(including leptospiral):
Gram stain and other microscopic techniques
Culture
Antimicrobial susceptibility
Antigen detection
Serotyping
Viral:
Culture
Antigen detection
Antibody levels
Genome analysis



Adapted from: *Guidelines for the collection of clinical specimens during field investigation of outbreaks*. Geneva, WHO, 2000 (WHO/CDS/CSR/EDC/2000.4).

APPENDIX 2: STEPS IN OUTBREAK MANAGEMENT

PREPARATION

- Health coordination meetings.
- Surveillance system – weekly health reports to WHO.
- Stockpiles – specimen kits, appropriate antibiotics, IV fluids.
- Epidemic investigation kits.
- Contingency plans for isolation wards in hospitals.
- Laboratory support.

DETECTION

If a certain number of cases of any of the following diseases/syndromes is diagnosed (i.e. alert threshold is passed):

- Acute watery diarrhoea in over-5-year-olds.
- Bloody diarrhoea.
- Suspected cholera.
- Measles.
- Meningitis.
- Acute haemorrhagic fever syndrome.
- Acute jaundice syndrome.
- Suspected polio (acute flaccid paralysis).
- Cluster of deaths of unknown origin.

(Diseases/syndromes in list should be modified according to the country's most updated epidemiological profile.
Inform your health coordinator as soon as possible. The health coordinator should inform the Ministry of Health and WHO.

RESPONSE

Confirmation

- The lead health agency should investigate reported cases to confirm the outbreak situation – number of cases higher than that expected for same period of year and population. Clinical specimens will be sent for testing.
- The lead health agency should activate an outbreak control team, with members drawn from relevant organizations: Ministry of Health, WHO and other United Nations organizations, nongovernmental organizations in the fields of health and water and sanitation, veterinary experts.

Investigation

- Confirm diagnosis (laboratory testing of samples).
- Define outbreak case definition.
- Count number of cases and determine size of population (to calculate attack rate).
- Collect/analyse descriptive data to date (e.g. time/date of onset, place/location of cases and individual characteristics such as age/sex).
- Follow up cases and contacts.
- Determine the at-risk population.
- Formulate hypothesis for pathogen/source/transmission.
- Conduct further investigation/epidemiological studies (e.g. to clarify mode of transmission, carrier, infectious dose required, better definition of risk factors for disease and at-risk groups.
- Write an investigation report (investigation results and recommendations for action).

Control

- Implement control measures specific for the disease and prevent exposure (e.g. isolation of cases in viral haemorrhagic fever outbreak).
- Prevent infection (e.g. immunization in measles outbreak).
- Treat cases as recommended in WHO guidelines.

EVALUATION

- Assess timeliness of outbreak detection and response, cost.
- Change public health policy if indicated (e.g. preparedness).
- Write outbreak report and disseminate.

APPENDIX 3: SAFE WATER AND SANITATION

The following are effective methods to obtain safe drinking-water:

Boiling
To make water safe for drinking and hygiene purposes, bring it to a vigorous, rolling boil and keep it boiling for 1 minute. This will kill, or inactivate, most of the organisms that cause diarrhoea.

Household filtration
Household filtration should considerably reduce the pathogens in the water. It should be followed by disinfection through chlorination or boiling.

Disinfection through chlorination
The following guidelines should be translated into messages that take into account locally available products and measuring devices. To make water safe by chlorination, the first step is to make a stock solution of chlorine.

A stock solution can be prepared by adding the following products to one litre of water:

Product (% **concentration** by **weight of available chlorine)**	**Amount for 1 litre**
Calcium hypochlorite (70%); or	15 g
Bleaching powder or chlorinated lime (30%); or	33 g
Sodium hypochlorite (5%); or	250 ml
Sodium hypochlorite (10%); or	110 ml

The stock solution must be stored in a closed container, in a cool dark place and used within 1 month. It should be used to prepare safe water as follows:

Stock solution	**Added volume** of water
0.6 ml or 3 drops	l litre
6 ml	10 litres
60 ml	100 litres

Mix by stirring, and allow the chlorinated water to stand for at least 30 minutes before using it. The free residual chlorine level after 30 minutes should be between 0.1 and 0.5 mg/litre. If the free residual chlorine is not within this range, the number of drops of the stock solution should be adjusted so that the final product falls within this range.

If the water is cloudy or turbid it must either be filtered before chlorination or boiled vigorously rather than chlorinated. Chlorination of turbid water might not make it safe.

Sanitation

Good sanitation can markedly reduce the risk of transmission of intestinal pathogens, especially where its absence may lead to contamination of clean water sources. High priority should be given to observing the basic principles of sanitary human waste disposal, as well as to ensuring the availability of safe water supplies.

Appropriate facilities for human waste disposal are a basic need of all communities; without such facilities there is a high risk of water-related diseases. Sanitary systems that are appropriate for the local conditions should be constructed with the cooperation of the community.

People will need to be taught how to use latrines, about the dangers of defecating on the ground, or in or near water, and about the importance of thorough hand-washing with soap or ash after any contact with excreta. The disposal of children's excreta in latrines needs to be emphasized.

See: - Franceys R, Pickford J, Reed R. *A guide to the development of on-site sanitation*. Geneva, WHO, 1992.

- Environmental health in emergencies and disasters: a practical guide
http://www.who.int/water_sanitation_health/hygiene/emergencies/emergencies2002/en/

- **Fact sheets on environmental sanitation WHO/EOS/96.4**
http://www.who.int/water_sanitation_health/hygiene/emergencies/envsanfactsheets/en/

APPENDIX 4: INJECTION SAFETY

Analysis of data collected as part of the Comparative Risk Assessment component of the Global Burden of Disease study suggests that the region that includes Sudan faces substantial challenges in terms of unsafe injection practices and transmission of blood-borne pathogens through injections. In this region, the proportion of new infections with hepatitis B, hepatitis C, and HIV that are attributable to unsafe injection practices are 58.3%, 81.7% and 7.1% respectively.

In Sudan, only 50% of EPI injections are administered safely (clean preparation, safe reconstitution and use of sterile syringe and needle), while therapeutic injections are safe in 30%. Sharps are presently collected in safety boxes in 130% of immunization and 0% of therapeutic settings, while they are found in open containers in 84% of health facilities.

Thus, in any relief efforts to assist the population and the displaced populations in this region of the world, safe and appropriate use of injections should be ensured through the following actions:

PATIENTS:

- State a preference for oral medications when visiting health care facilities.
- Demand a new, single-use syringe for every injection.

HEALTH WORKERS:

- Avoid prescribing injectable medication whenever possible.
- Use a new, single-use syringe for every injection.
- Do not recap syringes; discard them immediately in a sharps box to prevent needlestick injury.
- Dispose of by open-air incineration and burial of full sharps boxes.

IMMUNIZATION SERVICES:

- Deliver vaccines with matching quantities of auto-disable syringes and sharps boxes.
- Make sterile syringes and sharps boxes available in every health care facility.

ESSENTIAL DRUGS:

- Build rational use of injections into the national drug policy.
- Make single-use syringes available in quantities that match injectable drugs in every health care facility.

HIV-AIDS PREVENTION:

- Communicate the risk of HIV infection associated with unsafe injections.

HEALTH CARE SYSTEM:

- Monitor safety of injections as a critical quality indicator for health care delivery.

MINISTRY OF HEALTH:

- Coordinate safe and appropriate national policies with appropriate costing, budgeting and financing.

REMEMBER:

- Observe the "ONE SYRINGE, ONE NEEDLE SET, ONE INJECTION" rule
- A safe injection is one that:
  - Does no harm to the recipient.
  - Does not expose the health worker to avoidable risk.
  - Does not result in waste that puts other people at risk.
- An unsterile injection is usually caused by:
  - Reusable syringes that are not properly sterilized before use.
  - Single-use syringes that are used more than once.
  - Used syringes and needles that are not disposed of properly.

APPENDIX 5: KEY CONTACTS FOR SUDAN

Table 1: World Health Organization – Sudan

Office of the WHO Representative PO Box 2234 Khartoum – Sudan	**Dr Guido Sabatinelli** *The WHO Representative* (sabatinellig@sudanmail.net) **Location** Federal Ministry of Health Nile Avenue, Eastern Gate, Khartoum, Sudan Tel: +249 (11) 776 471 (office) +249 (11) 780 190 (direct) +249 (11) 781 707 Fax:+249 (11) 776 282 +873-382-420-336 (UNDP satellite in case of need) E-mail: whosud@sudanmail.net

Table 2: Relevant WHO Regional Offices and Headquarters Technical Staff

Area of work	EMRO contact	HQ contact
Communicable disease control in complex emergencies	Dr Ezzedine Mohsni mohsnie@emro.who.int	Dr Máire Connolly connollyma@who.int Dr Michelle Gayer gayerm@who.int Dr Pamela Mbabazi mbabazip@who.int
Outbreak alert and response	Dr Nadia Teleb telebn@emro.who.int	Dr Mike Ryan ryanm@who.int Dr Tom Grein greint@who.int Mr Pat Drury druryp@who.int
Acute lower respiratory infections	Dr Suzanne Farhoud farhouds@emro.who.int	Dr Shamim Qazi gazis@who.int
African trypanosomiasis		Dr Jean Jannin janninj@who.int
Bacillary dysentery – Cholera Typhoid Fever – Other Diarrhoeal diseases	Dr Nadia Teleb telebn@emro.who.int Dr Suzanne Farhoud farhouds@emro.who.int	Dr Claire-Lise Chaignat chaignatc@who.int
Diphtheria	Dr Ezzedine Mohsni mohsnie@emro.who.int	Dr Julian Bilous bilousj@who.int
Dracunculiasis		Dr Ahmed Tayeh tayeha@who.int
HIV/AIDS	Dr Jihane Tawilah tawilahj@emro.who.int Dr Hany Ziady ziadyh@emro.who.int	Dr Andrew Ball balla@who.int Dr Brian Pazvakavambwa pazvakavambwa@who.int

Leishmaniasis	Dr Riadh Ben-Ismail ismailr@emro.who.int	Dr François-Xavier Meslin meslinf@who.int
Leprosy		Dr Pannikar Vijaykumar pannikarv@who.int Dr Myo Thet Htoon htoonm@who.int
Lymphatic filariasis		Dr Francesco Rio riof@who.int Dr Sergio Yactayo yactayos@who.int
Malaria	Dr Hoda Atta attah@emro.who.int Dr Suzanne Farhoud farhouds@emro.who.int	Dr Aafje Rietveld rietvelda@who.int Dr Allan Schapira schapiraa@who.int
Measles	Dr Ezzedine Mohsni mohsnie@emro.who.int	Dr Brad Hersh hershb@who.int
Meningococcal disease	Dr Nadia Teleb telebn@emro.who.int	Dr William Perea peraw@who.int Dr. Eric Bertherat bertherate@who.int
Onchocerciasis		Dr Nevio Zagaria zagarian@who.int
Pertussis (whooping cough)	Dr Ezzedine Mohsni mohsnie@who.int	Dr Philippe Duclos duclosp@who.int
Poliomyelitis	Dr Faten Kamel kamelf@emro.who.int	Mr Chris Maher maherc@who.int Ms Claire Chauvin chauvinc@who.int
Rabies	Dr Riadh Ben-Ismail ismailr@emro.who.int	Dr François-Xavier Meslin meslinf@who.int
Schistosomiasis	Dr Riadh Ben-Ismail ismailr@emro.who.int	Dr Lorenzo Savioli saviolil@who.int Dr Dirk Engels engelsd@who.int
Soil-transmitted helminths	Dr Riadh Ben-Ismail ismailr@emro.who.int	Dr Lorenzo Savioli saviolil@who.int Dr Dirk Engels englesd@who.int

Tuberculosis	Dr Akihiro Seita seitaa@emro.who.int Dr Samiha Baghdadi baghdadis@emro.who.int	Dr Salah-Eddine Ottmani ottmanis@who.int Dr Malgosia Grzemska grzemska@who.int
Viral haemorrhagic fevers	Dr Nadia Teleb teleb@emro.who.int	Dr Cathy Roth rothc@who.int Mr. Pierre Formenty formentyp@who.int
Health aspects of biological agents		Dr Ottorino Cosivi cosivio@who.int
Injection safety	Dr Nadia Teleb telebn@who.int	Dr Yvan Hutin hutiny@who.int
Safe water	Dr Houssain Abouzaid abouzaidh@emro.who.int	Mr Jose Hueb huebj@who.int
Yellow fever		Dr. Sylvie Briand briands@who.int

APPENDIX 6: LIST OF WHO GUIDELINES ON COMMUNICABLE DISEASES

Title	Publication no./Date
FACT SHEETS	
Anthrax	Fact Sheet No. 264 October 2001 http://www.who.int/mediacentre/factsheets/fs264/en/
Cholera	Fact Sheet No. 107 Revised March 2000 http://www.who.int/mediacentre/factsheets/fs107/en
Dengue and dengue haemorrhagic fever	Fact Sheet No. 117 Revised April 2002 http://www.who.int/mediacentre/factsheets/fs117/en/
Diphtheria	Fact Sheet No. 89 Revised December 2000 http://www.who.int/mediacentre/factsheets/fs089/en/
Epidemic dysentery	Fact Sheet No. 108 (Being update)
Escherichia coli 0157:H7	Fact sheet No. 125 (Being update)
Food safety and foodborne illness	Fact Sheet No. 237 revised January 2002 http://www.who.int/mediacentre/factsheets/fs237/en/
Hepatitis B	Fact Sheet No. 204 Revised October 2000 http://www.who.int/mediacentre/factsheets/fs204/en/
Hepatitis C	Fact Sheet No. 164 Revised October 2000 http://www.who.int/mediacentre/factsheets/fs164/en/
Influenza	Fact Sheet No. 211 March 2003 http://www.who.int/mediacentre/factsheets/fs211/en/
Injection safety: background	Fact Sheet No. 231 Revised April 2002 http://www.who.int/mediacentre/factsheets/fs231/en/
Injection safety: facts & figures	Fact Sheet No. 232 (Being updated)
Injection safety: a Glossary	Fact Sheet No. 233 (Being updated)
Injection safety: questions & answers	Fact Sheet No. 234 (Being updated)
Malaria	Fact Sheet No. 94 http://www.who.int/mediacentre/factsheets/fs094/en/
Meales	Fact sheet N°286 http://www.who.int/mediacentre/factsheets/fs286/en/
Plague	Fact Sheet No. 267 February 2005 http://www.who.int/mediacentre/factsheets/fs267/en/
Poliomyelitis	Fact Sheet No. 114 Revised April 2003 http://www.who.int/mediacentre/factsheets/fs114/en/
Rabies	Fact Sheet No. 99 Revised June 2001 http://www.who.int/mediacentre/factsheets/fs099/en/

Salmonella	Fact Sheet No. 139 January 1997 (Being updated)
Smallpox	Smallpox http://www.who.int/mediacentre/factsheets/smallpox/en/
Tuberculosis	Fact Sheet No. 104 Revised March 2004 http://www.who.int/mediacentre/factsheets/fs104/en/
Typhoid fever and Paratyphoid fever	Water related diseases http://www.who.int/water_sanitation_health/diseases/typhoid/en/
The World Health Organization	About WHO http://www.who.int/about/en/

GUIDELINES/PUBLICATIONS/REPORTS	
Communicable Diseases control in emergencies - A field manual. http://www.who.int/infectious-disease-news/IDdocs/whocds200527/whocds200527chapters/index.htm	WHO/CDS/2005.27 ISBN 92 4 154616 6
Protocol for the assessment of national communicable disease surveillance and response systems. Guidelines for assessment teams http://www.who.int/emc-documents/surveillance/whocdscsrisr20012c.html	WHO/CDS/CSRIISR/2001.2 English only
Strengthening implementation of the Global Strategy for Dengue Fever/Dengue Haemorrhagic Fever Prevention and Control http://www.who.int/csr/resources/publications/dengue/en/whocdsdenic20001.pdf	WHO/CDS/(DEN)/IC/2000.1 English only
WHO report on global surveillance of epidemic-prone infectious diseases http://www.who.int/csr/resources/publications/surveillance/WHO_CDS_CSR_ISR_2000_1/en/	WHO/CDS/CSR/ISR/2000/1 English only
Guidelines for the collection of clinical specimens during field investigation of outbreaks http://www.who.int/emc-documents/surveillance/whocdscsredc2004c.html	WHO/CDS/CSR/EDC/2000.4 English only
Hepatitis A http://www.who.int/emc-documents/hepatitis/whocdscsredc20007c.html	WHO/CDS/EDC/2000.7 English only
Guidelines for epidemic preparedness and response to measles outbreaks http://www.who.int/emc-documents/measles/whocdscsrisr991c.html	WHO/CDS/CSR/ISR/99/1 English only
Influenza pandemic preparedness plan. The role of WHO and guidelines for national and regional planning http://www.who.int/csr/resources/publications/influenza/WHO_CDS_CSR_EDC_99_1/en/	WHO/CDS/CSR/EDC/99/1 English only
Plague manual: epidemiology, distribution, surveillance and control http://www.who.int/emc-documents/plague/whocdscsredc992c.html	WHO/CDS/CSR/EDC/99.2 English and French
Laboratory methods for the diagnosis of meningitis caused by Neisseria meningitidis, Streptococcus pneumoniae, and Haemophilus influenzae http://www.who.int/emc-documents/meningitis/whocdscsredc997c.html	WHO/CDS/CSR/EDC/99.7 English and French
Laboratory methods for the diagnosis of epidemic dysentery and cholera, 1999 http://www.cdc.gov/ncidod/dbmd/diseaseinfo/cholera/top.pdf	WHO/CDS/CSR/EDC//99.8 English and French
Control of epidemic meningococcal disease. WHO practical guidelines. 2nd ed. http://www.who.intlemc-documents/meningitis/whoemcbac983c.html	
Guidelines for the surveillance and control of anthrax in human and animals. 3rd ed.	
Cholera and other epidemic diarrhoeal diseases control. Technical cards on environmental sanitation, 1997 http://www.who.int/csr/resources/publications/cholera/WHO_EMC_DIS_97_6/en/	WHO/EMC/DIS/97/6

Epidemic diarrhoeal disease preparedness and response. Training and practice, 1998 (Participant's manual) http://www.who.int/emc-documents/cholera/whoemcdis973c.html	WHO/EMC/97.3 Rev.1 English, French and Spanish
Epidemic diarrhoeal disease preparedness and response. Training and practice, 1998 (Facilitator's guide) http://www.who.int/emc-documents/cholera/whoemcdis974c.html	WHO/EMC/97.4 Rev.1 English, French and Spanish
Dengue haemorrhagic fever: diagnosis, treatment, prevention and control. 2nd ed. http://www.who.int/csr/resources/publications/dengue/en/itoviii.pdf	1997 English only
Guidelines for the control of epidemics due to *Shigella dysenteriae* type 1 http://www.who.int/child-adolescent-health/Emergencies/Shigellosis_guidelines.pdf	Draft, 2005
VIDEOS	
Protecting ourselves and our communities from cholera (41 min). http://www.who.int/emc/diseases/cholera/videos.html	2000 English and French

WEB SITES	
WHO	http://www.who.int/
WHO/Cholera	http://www.who.int/topics/cholera/en/index.html
WHO Communicable Diseases and Surveillance	http://www.who.int/csr/en/
WHO Communicable Diseases Surveillance and Response	http://www.who.int/csr/
WHO Infectious Diseases news, documents and Communicable disease toolkits	http://www.who.int/infectious-disease-news/
WHO Roll Back Malaria partnership	http://www.rbm.who.int/
WHO/ Roll Back Malaria department	http://www.mosquito.who.int/malariacontrol
WHO/Stop TB	Http://www.stoptb.org/
WHO/Water and Sanitation	http://www.who.int/water_sanitation_health/en/

APPENDIX 7: IMMUNIZATION SCHEDULE FOR SUDAN

Vaccine	Schedule
BCG	Birth or 1st contact
DTP	6, 10, 14 weeks
OPV	Birth, 6, 10, 14 weeks
Measles	9 months
Vitamin A	6–59 months
TT	1st contact for WCBA*, 14th week of pregnancy, + 4–6 weeks

*WCBA = Women of childbearing age.

APPENDIX 8: MAP OF SUDAN

APPENDIX 9: POPULATION OF SUDAN, 2000

Northern and Central SUDAN			
State (wilayah)	**Capital**	**Area (km^2)**	**Population (2000)**
Blue Nile (An Nil al Azraq)	Ed Damazin	45 844	633 129
Gedaref (Al-Qadarif)	Gedaref	75 263	1 414 531
Gezira (Al-Jazirah)	Wad Medani	23 373	3 310 928
Kassala (Kassala)	Kassala	36 710	1 433 730
Khartoum (Al Khartum)	Khartoum	22 142	4 740 290
Northern Darfur (Shamal Darfur)	Al Fasher	296 420	1 409 894
Northern Kordofan (Shamal Kurdufun)	El Obeid	185 302	1 439 930
Northern State (Ash Shamaliyah)	Dongola	348 765	578 376
Red Sea (Al Bahr Al Ahmar)	Port Sudan	218 887	709 637
River Nile (Nahr an Nil)	Ed Damer	122 123	895 893
Sinnar (Sinnar)	Sinnar	37 844	1 132 758
Southern Darfur (Janub Darfur)	Nyala	127 300	2 708 007
Southern Kordofan (Janub Kurdufun)	Kadugli	79 470	1 066 117
Western Darfur (Gharb Darfur)	Geneina	79 460	1 531 682
Western Kordofan (Gharb Kurdufun)	Al Fula	111 373	1 078 330
White Nile (An Nil al Abyad)	Rabak	30 411	1 431 701
Subtotal		**1 840 687**	**25 514 933**
Southern SUDAN			
Bahr Al Jebel (Bahr-al-Jabal)	Juba	22 956	1 342 943
Eastern Equatoria (Sharq al Istiwa'iyah)	Kapoeta	82 542	1 234 486
Jongli (Junqali)	Bor	122 479	–
Lakes (Buheyrat)	Rumbek	40 235	–
Northern Bahr Al Ghazal (Shamal Bahr-al-Gazal)	Aweil	33 558	–
Unity State (Al Wahdah)	Bantiu	35 956	–
Upper Nile (A'ali an Nil)	Malakal	77 773	1 342 943
Warab	Warap	31 027	–
Western Bahr Al Ghazal (Gharb Bahr-al-Gazal)	Wau	93 900	–
Western Equatoria (Gharb al Istiwa'iyah)	Yambio	79 319	–
Subtotal		**619 745**	**3 920 372**
Grand total		**2 460 432**	**29 435 305**

Incomplete: population of southern Sudan is estimated at about 6 million.
(Data source: WHO/Sudan)

APPENDIX 10: BASIC HEALTH INDICATORS IN SUDAN

Life expectancy at birth (years)	55 (male) 58 (female) (2000)
Infant mortality rate	81 deaths per 1000 live births (2000)
Mortality rate for children aged <5 years	108 deaths per 1000 live births (2000)
Maternal mortality rate	500 deaths per 100 000 live births (1990–1998)
Population growth rate	2.4% (1980–2000)
Access to an improved water source	75% of population (2000)

(Data source: WHO/Sudan, 2004 and WHO/EMRO country profile – www.emro.who.int/sudan/).

WHO/CDS/2005.26

COMMUNICABLE DISEASE TOOLKIT

SUDAN

2. HEALTH SURVEILLANCE FORMS

World Health Organization

1. SUDAN SAMPLE MONTHLY MORBIDITY FORM

District: …… **Province/County: ……** **Town/Village/Camp**: **…….**

Health facility: …… **Supporting agency: ……** **Reporting period: From Monday ……/……/…….. To Sunday ……/……/…….**

Catchment population: …… **Under-5 population: ……** **Name of reporting officer: ……..**

	Week 1		Week 2		Week 3		Week 4		Total	
DISEASE / SYNDROME	**< 5**	**≥ 5**	**< 5**	**≥ 5**	**< 5**	**≥ 5**	**< 5**	**≥ 5**	**< 5**	**≥ 5**
*** Acute watery diarrhoea**										
*** Bloody diarrhoea**										
*** VHF – suspected**										
*** Measles**										
*** Meningitis – suspected**										
*** AFP (suspected poliomyelitis)**										
*** Yellow fever – suspected**										
ALRI / pneumonia										
Malaria – suspected										
Neonatal tetanus										
STIs										
Tuberculosis – suspected										
Fever of unknown origin										
Severe malnutrition (W/H <70%)										
Noncommunicable diseases										
Others										
TOTAL NUMBER OF CONSULTATIONS										

*** Diseases with outbreak potential – report as soon as possible to your district surveillance officer and district medical officer or health coordinator using outbreak alert form. See alert thresholds in "Surveillance system guidelines and alert thresholds" (*Annex 3*).**

For use by data management office: Form received: __/__/__ Validated □ Entered □ Record number: _

SUDAN SAMPLE MONTHLY MORTALITY FORM

District: …… **Province/County: ……** **Town/Village/Camp**: **…….**

Health facility: …… **Supporting agency: ……** **Reporting period: From Monday ……/……/…….. To Sunday ……/……/…….**

Catchment population: …… **Under-5 population: ……** **Name of reporting officer: ……..**

					Direct causes of death										Underlying causes of death				
No.	**First and middle names**	**Family name**	**Sex**	**Age (mth/ yr)**	**Fever**	**Watery diarrhoea #**	**Bloody diarrhoea**	**ALRI/pneumonia**	**Trauma/Injury**			**Specify cause or main symptoms if unknown**	**Unknown**	**Neonatal death §**	**Maternal death §**	**Malnutrition**	**Other (specify)**	**Date of death dd/mm/ yy**	**Location of death HF= health facility C = community**	**Lab. S= sample taken C= confirmed**
1																				
2																				
3																				
4																				
5																				
6																				
7																				
8																				
9																				
10																				

§ See case definitions list.

If this box is ticked, **also** specify cause in the "*specify cause*" column. Example: if cholera is suspected as the cause of the acute watery diarrhoea death, tick the acute watery diarrhoea column **and** write "cholera" in "*specify cause"* column.

For use by data management office: Form received: __/__/__ Validated □ Entered □ Record number:

3. SUDAN SAMPLE OUTBREAK ALERT FORM

District: …….. Province/County: ……

Town/Village/Camp: …….

Health facility: ……. Supporting agency: …….

Date: ……/……/…….

Name of reporting officer: ……..

Symptoms and signs: you can tick several boxes	Suspected disease/syndrome: tick one box only
☐ Acute watery diarrhoea	☐ Acute watery diarrhoea
☐ Bloody diarrhoea	☐ Bacillary dysentery/Shigellosis
☐ Fever	☐ Cholera
☐ Rash	☐ Measles
☐ Cough	☐ Meningitis
☐ Vomiting	☐ Malaria
☐ Neck stiffness	☐ VHF
☐ Jaundice	☐ Yellow fever
☐ Sore throat	☐ Poliomyelitis
☐ Bleeding	☐ Typhoid fever
☐ Acute paralysis or weakness	☐ Unknown disease
☐ Other: _____	☐ Other: _____
Total number of cases reported:	

Serial No.	Age	Sex	Location	Date of onset	Laboratory specimen taken (yes/no)	Treatment given	Outcome[a]	Final classification[b]

[a] Outcome: I = currently ill; R = Recovering or recovered; D = died.

[b] Final classification: S = suspected case with clinical diagnosis; C = confirmed case with laboratory diagnosis.

4. SUDAN SAMPLE OUTBREAK INVESTIGATION FORM

District: …….. **Province/County: ……**

Town/Village/Camp: …….

Health facility: …… **Supporting agency: …….**

Date: ……/……/…….

Name of reporting officer: ……..

1. PATIENT IDENTIFICATION

Case no: _____ **Name :_____**

Location in village or site: _____

Date of birth:___/___/_____ **Age:_____** **Sex: M F**

2. CLINICAL DATA

Date of onset of illness: ___/___/_____

- ☐ **Acute watery diarrhoea**
- ☐ **Bloody diarrhoea**
- ☐ **Fever**
- ☐ **Rash**
- ☐ **Cough**
- ☐ **Vomiting**
- ☐ **Neck stiffness**
- ☐ **Jaundice**
- ☐ **Sore throat**
- ☐ **Bleeding**
- ☐ **Acute paralysis or weakness**
- ☐ **Other: _____**

3. LABORATORY DATA

Sample: _____ **Date taken: ___/___/____** **Lab. received: ___/___/____**

Name of laboratory: _____

Type of test: _____ **Date of results: ___/___/___** **Result: Pos. Neg.**

4. FINAL CLASSIFICATION

Confirmed: ☐ **Laboratory** **Date of final diagnosis:___/___/**

☐ **Clinical case** **Discarded final diagnosis:_____**

5. FIELD INVESTIGATOR

Name: _____

Position: _____ **Signature: _____**

NOTE: ONE FORM PER CASE INVESTIGATED

WHO/CDS/2005.26

COMMUNICABLE DISEASE TOOLKIT

SUDAN

3. SURVEILLANCE SYSTEM GUIDELINES AND ALERT THRESHOLDS

World Health Organization

PURPOSE

These surveillance forms are for use in Sudan in the emergency phase. Included are: a weekly morbidity form, a weekly mortality form and a case-based reporting form for alerts. They aim to provide early warning of outbreaks of the following major communicable diseases:

– bacillary dysentery/shigellosis
– cholera
– hepatitis
– malaria
– measles
– meningococcal meningitis
– poliomyelitis
– typhoid fever
– viral haemorrhagic fever
– yellow fever.

In addition to the above outbreak-prone diseases, the main health problems are likely to be:

– endemic malaria
– malnutrition
– acute lower respiratory tract infection/pneumonia

An Early Warning System for Internal Displaced Population[1] example for malaria has also been included in this section of the toolkit (*see pages 5 - 9*).

REPORTING MECHANISMS

In each health facility, a daily register of consultations should be kept. The following is a suggested layout of the register:

OPD no.	**Date**	**Name**	**Location**	**Sex**	**DOB** <5 years	**DOB** >5 years	**New case/ Follow-up**	**Diagnosis**	**Treatment**	**Outcome**

One person in each health facility should be identified as responsible for data collection and notification of potential epidemics to the district surveillance officer or district medical officer. One person should be responsible for compiling the data from the daily register for the weekly morbidity report.

The monthly morbidity report should be filled out on a weekly basis from Saturday to Friday and compiled by the officer in-charge in a timely manner.

HOW TO FILL IN THE WEEKLY MORBIDITY REPORT

- Data should be recorded in two age categories – under 5 years and 5 years and older.

- Only new cases/first consultations should be reported; follow-up visits for the same disease should not be reported.

[1] WHO/MoH document dated 29 May 2004.

- All cases attending the health facility should be recorded, including those who are subsequently referred to hospital.

- At the end of each week, the reporting officer must count up all the cases and deaths from each disease as recorded in the outpatient and inpatient records. The health worker must select the primary diagnosis for the consultation, i.e. one disease/syndrome for each case.

- If one of the diseases is marked with an asterisk [*] on the form, that disease should be recorded as the main reason for consultation.

- **Diseases for immediate reporting** are marked with an asterisk [*] on the morbidity form. They must be reported immediately to the health coordinator or supervisor using the case-based reporting form used for reporting the specific disease.

- Other diseases/syndromes must be alerted to your health coordinator or supervisor when the **weekly alert thresholds** specified in the box are reached. If alert thresholds are passed, surveillance activities may need to be enhanced. If the number of cases of a disease/syndrome increases – such as in the event of an outbreak of meningitis or cholera, for example – active case-finding and case definitions may need to be revised.

HOW TO FILL IN THE WEEKLY MORTALITY FORM

This form is a line-listing of all deaths. Fill in all the details as required for each case, including name, age, sex, date and place of death, and record a main cause of death for each entry even if “unknown”.

Calculations of mortality rates can be performed as follows:

Crude mortality rate (CMR):

(total no. of deaths for the week / total population at the end of the week) x 10 000 persons = deaths/10 000 persons per week.

Under-5 mortality rate (U5MR):

(no. of deaths of children <5 years for the week/under-5 year population at the end of the week) x 10 000 persons = deaths/10 000 persons per week.

Alert thresholds for mortality are shown in the box below.

DISEASES/SYNDROMES FOR IMMEADIATE REPORTING

ALERT THRESHOLDS PER WEEK

Acute watery diarrhoea:	**5 cases in the 5 years and over age group (if cholera is suspected, one case is enough for reporting and further investigation)**
***Bloody diarrhoea:**	**5 cases or 1.5 times the baseline**
***Malaria:**	**1.5 times the baseline**
***Measles:**	**1 case**
***Meningitis - suspected:**	**5 cases or 1.5 times the baseline**
***VHF - suspected:**	**1 case**
***Yellow Fever - suspected:**	**1 case**
***AFP (suspected poliomyelitis):**	**1 case**
***Neonatal tetanus:**	**1 case**
Fever of unknown origin:	**1.5 times the baseline**
Severe malnutrition:	**3 cases**

Baseline = average weekly number of cases of the disease calculated over the last 3 weeks.

Alert thresholds for deaths in displaced populations

⇒**CMR is >1/10,000/day**

⇒**U5MR is >2/10,000/day**

Use Alert form to report to District Surveillance Officer and district health authorities if one of these thresholds is reached in a week

Diseases for immediate reporting *are marked with an asterisk [*] on the morbidity form. They must be reported immediately to the health coordinator or supervisor using a case-based reporting form used for reporting the specific disease.*

Darfur humanitarian crisis - Early Warning System for malaria epidemics in the Internal displaced population (IDP)[2].

Alert threshold:
Clustering of malaria referrals/inpatients and deaths, especially among older children and adults, will require immediate investigation (within 24-48 hours) to determine the cause, effect and the potential magnitude of the epidemic. Control measures, notably improved access to free diagnosis and treatment with artemisinin based combination therapy (ACT), must be implemented immediately (within one week) if a falciparum malaria epidemic is confirmed.

Declaring an epidemic over:
The epidemic is declared over when a steady decline over a period of 4-6 weeks occurs, and the number of malaria referrals fall below the levels of the time the alert is given.

Suspected disease or condition	Diagnostic test	Specimen	When to collect	How to prepare, store and ship	Results
Malaria **REFERENCES:** "Basic Laboratory Methods in Medical Parasitology" WHO, Geneva, 1991 "Malaria rapid diagnosis - making it work" (WHO, 2003), http:www.rbm.who.int	Presence of malarial parasites in thick or thin blood film OR Positive malaria rapid diagnostic test	Blood Usually finger-stick sample	To confirm diagnosis in suspected malaria cases; for patient management in severe malaria admissions; for follow up of suspected treatment failures (microscopy only)., If case loads are too high to allow testing of all suspected patients, test a percentage of cases to monitor the slide/test positivity rate.	Blood smear: Collect blood directly onto correctly cleaned and labeled microscope slides and prepare thick and thin smears. • Allow smears to dry thoroughly. • Stain using the appropriate stain (Giemsa stain) and technique. • Store stained and thoroughly dried slides at room temperature out of direct sunlight. Rapid diagnostic test: according to manufacturer's instructions. Rapid diagnositic test should be stored and transported in a "cool chain". Temperature stability data should be requested from manufacturer before purchase.	Thick and thin smear results should be available within 1 hour of preparation. Microscopic examination of malarial slides may also reveal the presence of other blood-borne parasites.

[2] WHO/MoH document dated 29 May 2004.

CONFIRMING MALARIA EPIDEMICS IN POPULATIONS WHERE NO HISTORIC RECORDS ARE AVAILABLE FOR COMPARISON[3]

(IDP POPULATIONS, DARFUR 2004)

1. Facilities with outpatient department only: Confirmation of a malaria epidemic can be done using a comparison of malaria case numbers (suspected and confirmed) by age grouping (children under 5 years, as compared to older children and adults) for the past 4-8 weeks.

An epidemic is declared when:

- total fever cases are increasing and the proportion of affected older children and adults is increasing or
- when in older children and adults the proportion of fever cases that are confirmed as malaria is steadily increasing in recent weeks despite the ratio of confirmed to suspected cases has not changed much in young children.

Examples are given below:

- Chart 1.1 : the total fever cases (clinical malaria cases), split by age grouping
- Chart 1.2 : the ratio of confirmed cases to fever cases in children under 5
- Chart 1.3 : the ratio of confirmed cases to fever cases in older children and adults

Rapid diagnostic tests can be used to confirm that the upward trend is due to malaria infection.

Example 1: Confirming malaria epidemics - facilities with outpatient department only

Age group	under 5 years		5 years and over	
Diagnosis	**all fever cases**	**Confirmed malaria**	**all fever cases**	**Confirmed malaria**
8 weeks ago	96	80	10	5
7 weeks ago	84	68	32	18
6 weeks ago	102	88	42	20
5 weeks ago	67	59	53	22
4 weeks ago	87	80	41	29
3 weeks ago	100	87	39	28
2 weeks ago	150	140	82	68
Last week	170	156	98	89

[3] Source: Field Guide for Malaria Epidemic Reporting and Assessment, WHO/2004, http://www.rbm.who.int

Chart 1.1: Fever cases by age group

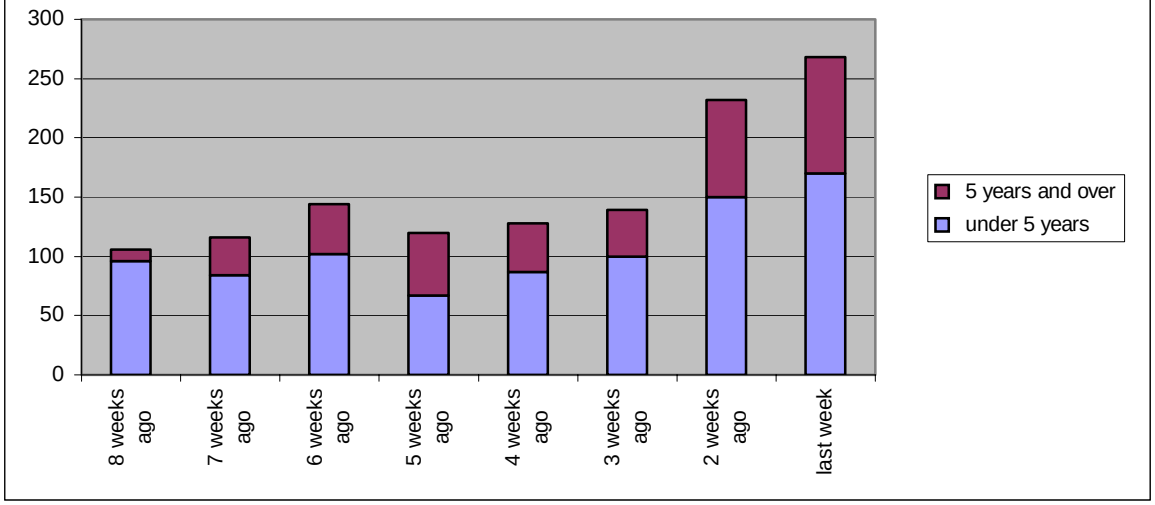
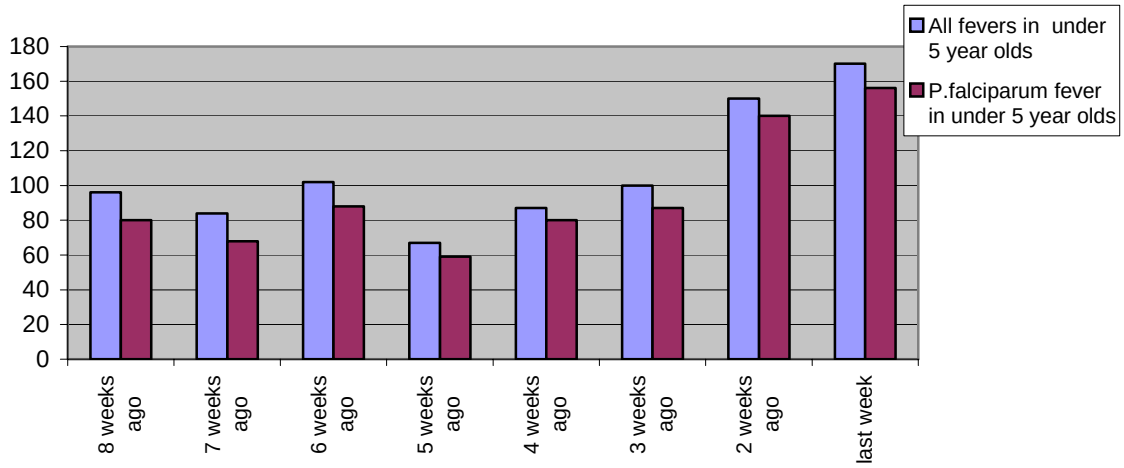
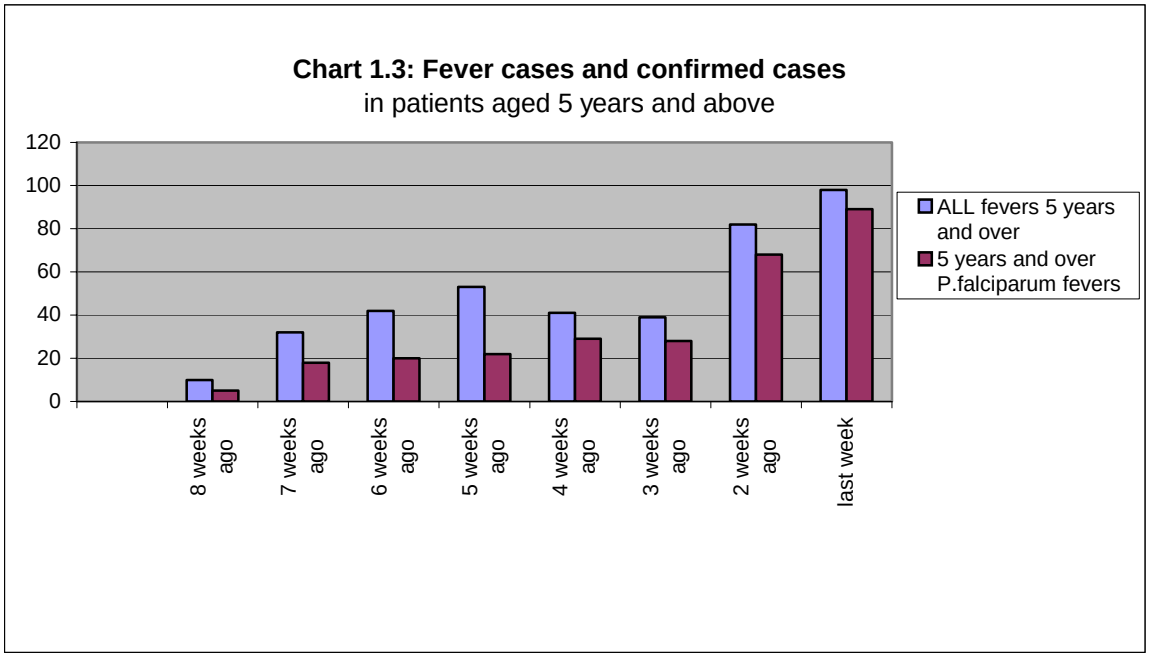


Chart 1.2: Fever cases and confirmed cases
in children under 5 years of age





2. Facilities with inpatient department: conformation of a malaria epidemic can be done by comparing malaria hospital admissions, deaths and case-fatality rates by age grouping (children under 5 vs. older children and adults) for the past 4-8 weeks. During an epidemic the graphs generated using in-patient data show an obvious upward trend in hospital admissions and deaths attributed to malaria.

Examples are given below:

- Chart 2.1: hospital admissions and deaths attributed to malaria in children under 5 years.
- Chart 2.2: hospital admissions and deaths attributed to malaria in older children and adults.
- Chart 2.3: malaria deaths divided by malaria admissions (inpatient case fatality rate).The graph of the case-fatality rate (deaths/admitted) shows a sharp upward trend in the most recent weeks.

Example 2: Confirming malaria epidemics - facilities with inpatient department

Children <5: hospital malaria data			
	admitted	**deaths**	**CFR* (%)**
8 weeks ago	56	5	8.9
7 weeks ago	49	3	6.1
6 weeks ago	63	5	7.9
5 weeks ago	55	4	7.3
4 weeks ago	70	6	8.6
3 weeks ago	68	5	7.4
2 weeks ago	73	10	13.7
last week	80	13	16.3

*CFR - case fatality rate

Chart 2.1: Hospital admissions and deaths attributed to malaria
in children under 5 years of age

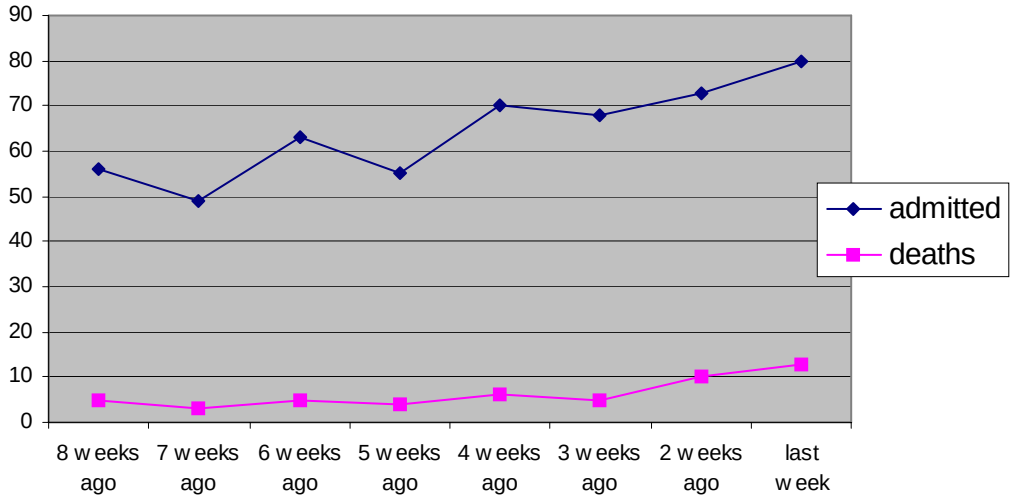
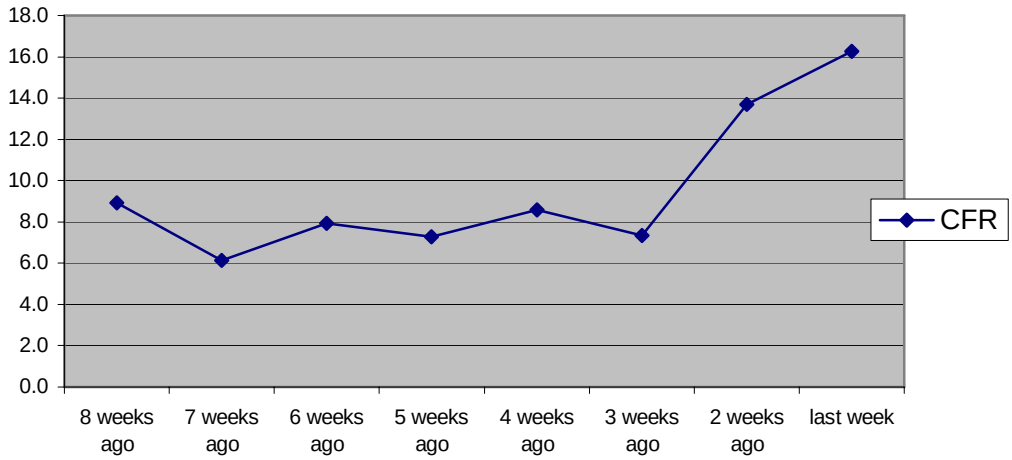


Chart 2.2: Hospital case-fatality rate (%)
in children under 5 years of age



Similar graphs can be made for older children and adults.

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COMMUNICABLE DISEASE TOOLKIT

SUDAN

4. CASE DEFINITIONS

World Health Organization

WHO/FMOH-RECOMMENDED CASE DEFINITIONS

ACUTE WATERY DIARRHOEA

Any person with 3 or more abnormally loose or fluid stools in the past 24 hours **with or without** dehydration.

Suspected cholera case:

- Person aged >5 years with severe dehydration or death from acute watery diarrhoea with or without vomiting.
- Person aged >2 years with acute watery diarrhoea in an area where there is a cholera outbreak.

Confirmed cholera case:
Isolation of *Vibrio cholera* O1 or O139 from diarrhoeal stool sample.

ACUTE BLOODY DIARRHOEA

Person with acute diarrhoea with visible blood in the stool.

Suspected shigellosis case:
Any person with acute diarrhoea, visible blood in the stool and fever.

Confirmed shigellosis case:
Isolation of *Shigella dysenteriae* type 1 through stool culture and serology from a suspected case.

ACUTE HAEMORRHAGIC FEVER SYNDROME

Any person with severe illness, acute onset of fever **and** at least one of the following:

– sore throat (found in Lassa fever only)
– bloody stools
– vomiting blood
– unexplained bleeding from any other site (gums, nose, vagina, skin, eyes).

ACUTE JAUNDICE SYNDROME (INCLUDING YELLOW FEVER)

Any person with acute onset of jaundice **with or without** fever **and** absence of any known precipitating factors.

Confirmed yellow fever case:
Presence of yellow fever-specific IgM or a fourfold or greater increase in serum IgG levels between the acute and convalescent serum samples. Yellow fever can also be confirmed by isolation of the yellow fever virus in blood or detection of yellow fever antigen in tissues by immunohistochemistry.

MEASLES

Any person with fever **and** maculopapular (i.e. non-vesicular) rash **and** cough, coryza (i.e. runny nose) or conjunctivitis (i.e. red eyes)

or

Any person in whom a clinical health worker suspects measles infection.

Confirmed measles case:
A case that meets the case definition and is laboratory-confirmed through serology (presence of measles-specific IgM antibodies) or linked epidemiologically to a laboratory-confirmed case.

MENINGITIS

Suspected meningitis case:
Any person with sudden onset of fever (>38 °C axillary) **and** one of the following:

- neck stiffness
- altered consciousness
- other meningeal sign **or** petechial/purpural rash.

In children aged <1 year, meningitis is suspected when fever is accompanied by a bulging fontanelle.

Confirmed meningitis case:

A suspected case with laboratory confirmation through positive cerebrospinal fluid antigen detection **or** positive cerebrospinal fluid culture **or** positive blood culture.

ACUTE FLACCID PARALYSIS (SUSPECTED POLIOMYELITIS)

Acute flaccid paralysis (AFP) in a child aged <15 years, including Guillain - Barré syndrome **or** any paralytic illness in a person of any age.

Confirmed case:
An AFP case with laboratory-confirmed wild poliovirus in stool sample.

ACUTE LOWER RESPIRATORY TRACT INFECTION / PNEUMONIA IN CHILDREN AGED UNDER 5 YEARS

Cough or difficult breathing

and

Breathing 50 or more times per minute for infants aged 2 months to 1 year

Breathing 40 or more times per minute for children aged 1 to 5 years

and

No chest indrawing, no stridor, no general danger signs.

Note: **Severe pneumonia** = cough or difficult breathing **plus** any general danger sign (unable to drink or breastfeed, vomits everything, convulsions, lethargic or unconscious) or chest indrawing or stridor in a calm child.

MALARIA

Uncomplicated malaria
Patient with fever or history of fever within the past 48 hours (with or without other symptoms such as nausea, vomiting and diarrhoea, headache, back pain, chills and myalgia)

Severe malaria
Patient with symptoms as for uncomplicated malaria, plus drowsiness with extreme weakness and associated signs and symptoms related to organ failure (e.g. disorientation, loss of consciousness, convulsions, severe anaemia, jaundice, haemoglobinuria, spontaneous bleeding, pulmonary oedema and shock).

Confirmed malaria case (uncomplicated or severe):
Patient with uncomplicated or severe malaria with laboratory confirmation of diagnosis by malaria blood film or other diagnostic test for malaria parasites.

NEONATAL TETANUS

Suspected case:
Any neonatal death between 3 and 28 days of age in which the cause of death is unknown

or

Any neonate reported as having suffered from neonatal tetanus between 3 and 28 days of age but not investigated.

Confirmed case:

Any neonate with normal ability to suck and cry during the first 2 days of life but who, between 3 and 28 days of age, can no longer suck normally and becomes stiff or has convulsions (i.e. jerking of the muscles) or both.

Hospital-reported cases are considered as confirmed cases.

The diagnosis is entirely clinical and does not depend on bacteriological confirmation.

SEXUALLY TRANSMITTED INFECTIONS

Genital ulcer syndrome
Ulcer on penis or scrotum in men and on labia, vagina or cervix in women, with or without inguinal adenopathy.

Urethral discharge syndrome
Urethral discharge in men with or without dysuria.

Vaginal discharge syndrome
Abnormal vaginal discharge (amount, colour and odour), with or without lower abdominal pain or specific symptoms or specific risk factors.

Lower abdominal pain
Lower abdominal pain and pain during sexual relations, with examination showing vaginal discharge, lower abdominal tenderness on palpation or axillary temperature >38 °C.

TUBERCULOSIS

Suspected TB case:
Any person who presents with symptoms or signs suggestive of pulmonary TB, in particular cough of long duration (>2 weeks).

May also be coughing blood, have chest pain, shortness of breath, fever/night sweats, tiredness, loss of appetite and significant weight loss.

All TB suspects should have three sputum samples examined by light microscopy. Early morning samples are more likely to contain the TB organism than samples taken later in the day.

Pulmonary TB smear-positive (PTB+)
Diagnostic criteria should include:

- At least two sputum smear specimens positive for acid-fast bacilli (AFB)
  or
- One sputum smear specimen positive for AFB and radiographic abnormalities consistent with active pulmonary TB
  or
- One sputum smear specimen positive for AFB and a culture positive for *M. tuberculosis*.

Pulmonary TB smear-negative (PTB–)
A case of pulmonary tuberculosis that does not meet the above definition for smear-positive TB. Diagnostic criteria should include:

- At least three sputum smear specimens negative for AFB
  and
- Radiographic abnormalities consistent with active pulmonary TB
  and
- No response to a course of broad-spectrum antibiotics
  and
- Decision by a clinician to treat with a full course of anti-TB chemotherapy.

FEVER OF UNKNOWN ORIGIN

Any person with fever (>38 °C axillary) in whom all obvious causes of fever have been excluded.

SEVERE MALNUTRITION

In children aged 6–59 months (65–110 cm in height):

- Weight-for-height (W/H) index < –3 Z-scores (on table of NCHS/WHO normalized reference values of weight-for-height by sex) (<70% of normal)
  or
- Bilateral pitting oedema irrespective of W/H, in absence of other causes.

TRAUMA/INJURY

Any person who has sustained, either directly or indirectly, a fatal or non-fatal injury caused by:

- war: any weapons or explosion of a landmine or other unexploded ordnance (UXO).
- other: road traffic accidents, domestic violence, burns.

Note: Landmine injuries relate to buried mines (e.g. antipersonnel and/or antivehicle mines). UXO injuries arise from explosive objects/devices that are typically above ground at the time of detonation, such as cluster munitions that did not detonate on impact.

MATERNAL DEATH

Death of a woman while pregnant or within 42 days of termination of pregnancy, regardless of the site or duration of pregnancy, from any cause related to or aggravated by the pregnancy or its management.

NEONATAL DEATH

Death of a liveborn infant during the first 28 days of life. It is a classification by age, not cause.

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COMMUNICABLE DISEASE TOOLKIT

SUDAN

5. GUIDELINES FOR OUTBREAK CONTROL

World Health Organization

TABLE 1. STEPS IN MANAGEMENT OF AN OUTBREAK

1. PREPARATION

- Health coordination meetings.
- Surveillance system – weekly health reports to WHO.
- Stockpiles – specimen kits, appropriate antibiotics, IV fluids.
- Epidemic investigation kits.
- Contingency plans for isolation wards in hospitals.
- Laboratory support.

2. DETECTION

If a certain number of cases of any of the following diseases/syndromes is diagnosed (i.e. alert threshold is passed):

- acute watery diarrhoea.
- bloody diarrhoea.
- suspected cholera.
- measles.
- meningitis.
- acute haemorrhagic fever syndrome.
- acute jaundice syndrome.
- acute flaccid paralysis (suspected poliomyelitis).
- a cluster of deaths of unknown origin.

(Diseases/syndromes in list to be modified according to country profile).

Inform your health coordinator as soon as possible. The health coordinator should inform the Ministry of Health and WHO.

3. RESPONSE

Confirmation

- The lead health agency should investigate reported cases to confirm the outbreak situation – number of cases higher than expected for same period of year and population. Clinical specimens will be sent for testing.
- The lead health agency should activate an outbreak control team with membership from relevant organizations: Ministry of Health, WHO and other United Nations organizations, nongovernmental organizations in the fields of health and water and sanitation, veterinary experts.

Investigation

- Confirm diagnosis (laboratory testing of samples).
- Define outbreak case definition.
- Count number of cases and determine size of population (to calculate attack rate).
- Collect/analyse descriptive data to date (e.g. time/date of onset, place/location of cases and individual characteristics such as age/sex).
- Follow up cases and contacts.
- Determine the at-risk population.
- Formulate hypothesis for pathogen/source/transmission.
- Conduct further investigation/epidemiological studies (e.g. to clarify mode of transmission, carrier, infectious dose required, better definition of risk factors for disease and at-risk groups).
- Write an investigation report (investigation results and recommendations for action).

Control

- Implement control measures specific for the disease and prevent exposure (e.g. isolation of cases in viral haemorrhagic fever outbreak).
- Prevent infection (e.g. immunization in measles outbreak).
- Treat cases as recommended in WHO guidelines.

4. EVALUATION

- Assess timeliness of outbreak detection and response, cost.
- Change public health policy if indicated (e.g. preparedness).
- Write outbreak report and disseminate.

TABLE 2. RESOURCES NEEDED FOR OUTBREAK RESPONSE

- Personnel (trained staff)
- Supplies (e.g. oral rehydration salts, intravenous fluids, water containers, water purifying tablets, drinking cups, vaccines, vitamin A, monitoring forms, vaccination cards, tally sheets)
- Treatment facilities (location, beds available, stocks of basic medical supplies)
- Laboratory facilities (location, capacity, stocks of reagents, etc.)
- Transport (sources of emergency transport and fuel, cold chain)
- Communication links (between health centres; between Ministry of Health, nongovernmental organizations and United Nations agencies)
- Computers
- In an outbreak requiring an immunization campaign:
  - safe injection equipment (e.g. auto-destruct syringes and safety boxes (puncture-resistant boxes),
  - immunization facilities (location, capacity),
  - cold-chain equipment (number and condition of refrigerators, cold-boxes, vaccine carriers, ice-packs).

TABLE 3. RISK FACTORS FOR OUTBREAKS IN EMERGENCY SITUATIONS

Acute respiratory infections	Inadequate shelter with poor ventilation
	Indoor cooking, poor health care services
	Malnutrition, overcrowding
	Age group under 1 year old
	Large numbers of elderly
	Cold weather
Diarrhoeal diseases	Overcrowding
	Inadequate quantity and/or quality of water
	Poor personal hygiene
	Poor washing facilities
	Poor sanitation
	Insufficient soap
	Inadequate cooking facilities
Malaria	Mass population movement with increased vulnerability of displaced populations because of malnutrition, concomitant diseases, settlement in marginal areas close to mosquito breeding sites, housing in temporary shelters with increased exposure to mosquito bites, increased population density promoting malaria transmission.
	Poor access to health care (curative and preventive), combined with breakdown of health services, existing health facilities overwhelmed.
	Interruption of vector control activities
	Environmental degradation encouraging vector breeding
Measles	Measles immunization coverage rates below 80% in country of origin
	Population movement
	Overcrowding
Meningococcal meningitis	Meningitis belt
	Dry season
	Dust storms
	Overcrowding
	High rates of acute respiratory infections

TABLE 3 (*continued*)

Viral haemorrhagic fever	Lack of hygiene, poor sanitation, contact with objects/food contaminated with rodent excreta; unsafe food handling and storage practices (Lassa fever) Population displacement with subsequent overcrowding Poor access to health services, poor isolation and protection measures (barrier nursing) Tick-infested areas (Crimean–Congo haemorrhagic fever) Handling or eating ill or dead infected primates (Ebola) or rodents (Lassa fever)
Yellow fever	Unvaccinated people moving to areas of endemicity are at risk. Overcrowding Open water storage provides favourable habitat for *Ae. aegypti* Old tyres, old water containers increase vector breeding sites Poor drainage leading to pools and open channels of water) may increase vector breeding opportunities.

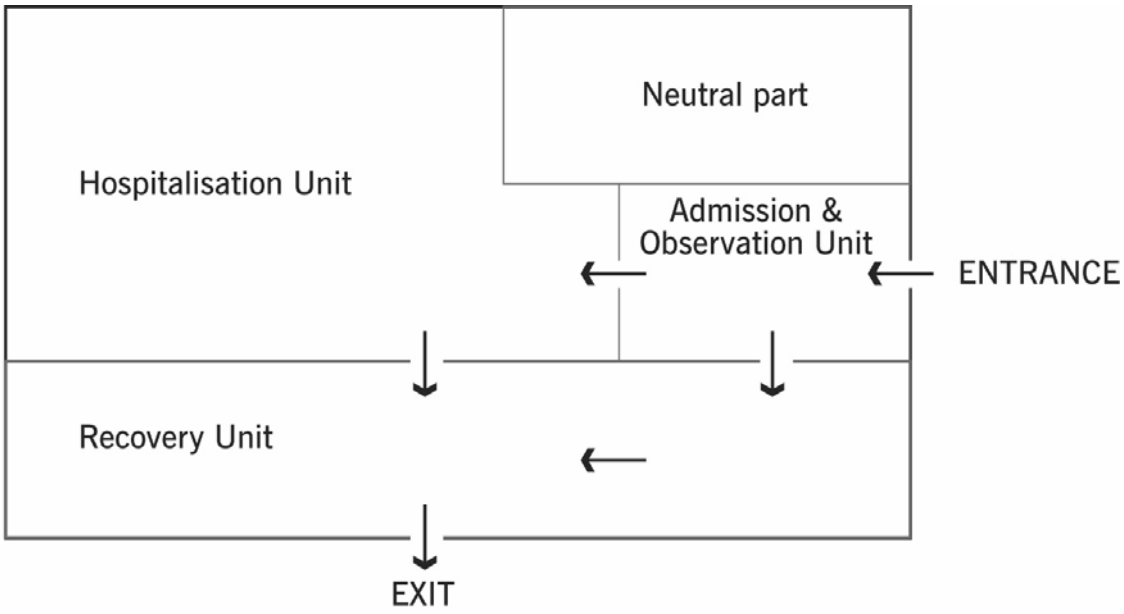
TABLE 4. ESSENTIAL HYGIENE RULES IN A CHOLERA TREATMENT CENTRE

Mode of transmission	Essential rules in the unit	Additional recommended rules
People	• Access limited to patient + one family member + staff • One-way flow of people	• Ideally, only one carer per patient • Three separate spaces within unit (see Figure 1)
Water	• Safe water (chlorination concentration according to specific use; see *Table 5*) • Large quantity needed (minimum 10 litres/person per day)	• Ideally 50 litres/patient per day
Hands	• Hand-washing stations with safe water and soap in sufficient quantities • Wash hands with water and soap – before and after taking care of patients – after using the latrines – before cooking or eating – after leaving the admission ward	• Cut and clean nails
Food	• Cooked food • Health care workers should not handle food or water	• Food provided by the unit (preferably not by families) • Large stocks of food may be "tempting "and may lead to security problems
Clothes	• Wash clothes and linen with the appropriate chlorine solution	• If no chlorine available, wash clothes with soap and dry them in the sun
Environmental contamination (faeces and waste)	• Ensure exclusive latrines for the unit • Disinfect buckets, soiled surfaces and latrines regularly with the appropriate chlorine solution (see *Table 5*) • Incinerator for medical waste	• Latrines at least 100 metres away from wells or surface sources • Special cholera beds
Corpses	• Separate morgue • Disinfect corpses (see *Table 5*)	• Find ways to have safe burial practices • Bury corpses as soon as possible

Developed by the WHO Global Task Force on Cholera Control.

Figure 1:

Organization of an emergency treatment centre and patient-flow



Four **separate** spaces:

- Admission and observation unit
- Neutral part: staff office and staff rest room, hospital kitchen, store rooms
- Hospitalisation unit: reserved for severe patients with IV fluids
- Recovery unit: oral rehydration space

In each space. Ensure exclusive latrines, washing areas, large quantity of water and safe disposal of waste

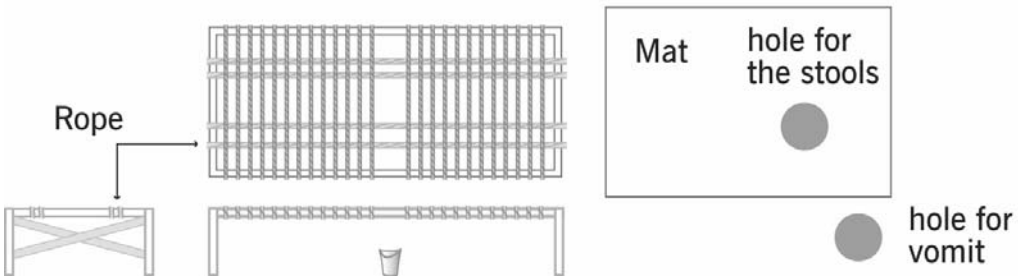


TABLE 5. PREPARATION AND USE OF DISINFECTANTS

Starting with	**2% solution**	**0.2% solution**	**0.05% solution**
Calcium hypochlorite at 70% active chlorine ("high-test hypochlorite", "HTH")	30 g/litre or 2 tablespoons/litre	30 g/10 litres or 2 tablespoons/10 litres	7 g/10 litres or ½ tablespoon/10 litres
Chlorinated lime at 30% active chlorine ("bleaching powder")	66 g/litre or 4 tablespoons/litre	66 g/10 litres or 4 tablespoons/10 litres	16 g/10 litres or 1 tablespoon/10 litres
Sodium hypochlorite solution at 6% active chlorine ("household bleach")	333 ml/litre or 22 tablespoons/litre	333 ml/10 litres or 22 tablespoons/10 litres	83 ml/10 litres or 5 tablespoons/10 litres
USE FOR DISINFECTION OF	Excreta Corpses Shoes	Floor Utensils Beds	Hands Skin Clothes

Developed by the WHO Global Task Force on Cholera Control.

Approximate measurements:
1 teaspoon = 5 ml
1 tablespoon = 15 ml, or 3 teaspoons

Do not use metallic buckets for preparation and storage of chlorinated solutions.

TABLE 6. CHOLERA TREATMENT SUPPLIES PER POPULATION

How to estimate the initial amount of supplies needed for a cholera outbreak:
(0.2% of the population expected to fall ill initially).

The table below gives an estimate of the amount of supplies you will need according to the number of people in your area. To find the amounts needed for each item, look in the column under the approximate population of your catchment area (to the nearest 5000). You may add several columns (e.g. if your health facility serves 35 000 people, add the amounts in the 10 000 and 5000 columns to those in the 20 000 column). Write the amount needed at your health facility in the empty column on the right.

On the basis of drug resistance in your area, choose only one of the antibiotics.

Item	**Population** (+ *numbers expected to fall ill)*						Your area
	5000 *(10)*	10 000 *(20)*	15 000 *(30)*	20 000 *(40)*	50 000 *(100)*	100 000 *(200)*	
Rehydration supplies							
ORS packets (for 1 litre each)	65	130	195	260	650	1300	
Nasogastric tubes (adults) 5.3/3.5 mm (16 Flack) 50 cm	1	1	1	2	3	6	
Nasogastric tubes (children)	1	1	1	2	3	6	
Ringer's lactate bags, 1 litre, with giving sets	12	24	36	48	120	240	
Scalp vein sets	2	3	4	5	10	20	
Antibiotics							
Doxycycline, 100 mg (adults)	6	12	18	24	60	120	
Erythromycin 250 mg (children)	24	48	72	96	240	480	
Other treatment supplies							
Large water dispensers with tap (marked at 5–10 litres)	1	1	1	2	2	4	
1-litre bottles for ORS solution	2	4	6	12	20	40	
0.5-litre bottles for ORS solution	2	4	6	12	20	20	
Tumblers, 200 ml	4	8	12	16	40	80	
Teaspoons	2	4	6	8	20	40	
Cotton wool, kg	1/2	1	11/2	2	5	10	
Adhesive tape, reels	1	1	1	2	3	6	

Developed by the WHO Global Task Force on Cholera Control.

TABLE 7. DYSENTERY TREATMENT SUPPLIES PER POPULATION

How to estimate the amount of supplies needed for a dysentery outbreak:
(0.2% of the population expected to fall ill initially).

The table below gives an estimate of the amount of supplies you will need according to the number of people in your area. To find the amounts needed for each item, look in the column under the approximate population of your catchment area (to the nearest 5000). You may add several columns (e.g. if your health facility serves 35 000 people, add the amounts in the 10 000 and 5000 columns to those in the 20 000 column). Write the amount needed at your health facility in the empty column on the right.

On the basis of drug resistance in your area, choose only one of the antibiotics.

Item	**Population** (+ *numbers expected to fall ill)*						Your area
	5000 *(10)*	10 000 *(20)*	15 000 *(30)*	20 000 *(40)*	50 000 *(100)*	100 000 *(200)*	
Rehydration supplies							
ORS packets (for 1 litre each)	10	20	30	40	100	200	
Ringer's lactate bags, 1 litre, with giving sets	2	4	6	8	20	40	
Scalp vein sets	1	1	2	2	5	10	
Antibiotics							
Ciprofloxacin, 500 mg	100	200	300	400	1000	2000	
Other treatment supplies							
Large water dispensers with tap (marked at 5–10 litres)	1	1	1	1	1	2	
1-litre bottles for ORS solution	1	1	2	2	5	10	
0.5-litre bottles for ORS solution	1	1	2	2	5	10	
Tumblers, 200 ml	1	2	3	4	10	20	
Teaspoons	1	1	2	2	5	10	
Cotton wool, kg	1/2	1	1 1/2	2	5	10	
Adhesive tape, reels	1	1	1	2	3	6	
Hand soap, kg	2	4	6	8	20	40	
Boxes of soap for washing clothes	3	6	9	12	30	60	
1-litre bottle of cleaning solution (2% chlorine or 1–2% phenol)	1	1	1	1	2	4	

Developed by the WHO Global Task Force on Cholera Control.

TABLE 8. TYPHOID FEVER TREATMENT SUPPLIES PER POPULATION

How to estimate the amount of supplies needed for a typhoid outbreak:
(0.2% of the population expected to fall ill initially).

The table below gives an estimate of the amount of supplies you will need according to the number of people in your area. To find the amounts needed for each item, look in the column under the approximate population of your catchment area (to the nearest 5000). You may add several columns (e.g. if your health facility serves 35 000 people, add the amounts in the 10 000 and 5000 columns to those in the 20 000 column). Write the amount needed at your health facility in the empty column on the right.

On the basis of drug resistance in your area, choose only one of the antibiotics.

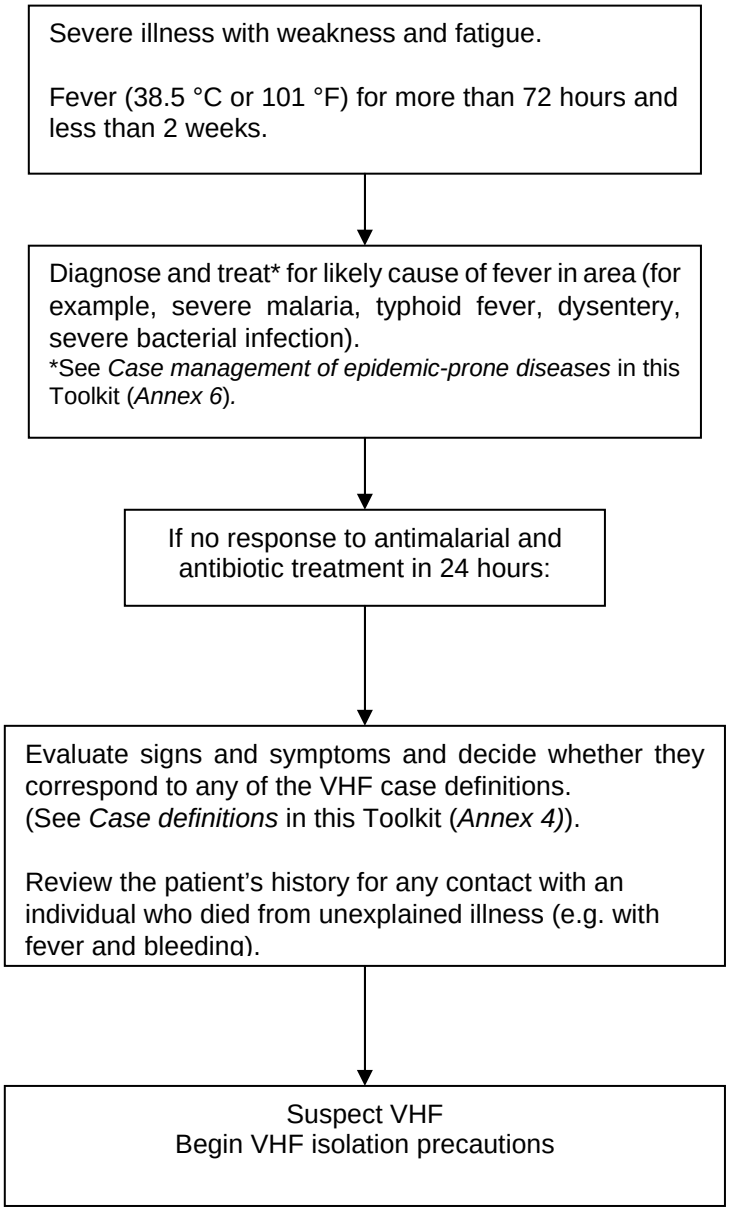
Item	**Population** (+ *numbers expected to fall ill)*						Your area
	5000 *(10)*	10 000 *(20)*	15 000 *(30)*	20 000 *(40)*	50 000 *(100)*	100 000 *(200)*	
Rehydration supplies							
ORS packets (for 1 litre each)	**10**	**20**	**30**	**40**	**100**	**200**	
Ringer's lactate bags[a], 1 litre, with giving sets	1	2	3	4	10	20	
Scalp vein sets	1	1	2	2	5	10	
Antibiotics							
Chloramphenicol, 250 mg	2500	5000	7500	10 000	25 000	50 000	
Amoxicillin, 500 mg	1680	3360	5040	6 720	16 800	33 600	
Co-trimoxazole, (SMX 400 mg + TMP 80 mg)	840	1680	2520	3 360	8 400	16 800	
Cefixime, 200 mg[b]	840	1680	2520	3 360	8 400	16 800	
Other treatment supplies							
Large water dispensers with tap (marked at 5–10 litres)	1	1	1	1	1	2	
1-litre bottles for ORS solution	1	1	2	2	5	10	
0.5-litre bottles for ORS solution	1	1	2	2	5	10	
Tumblers, 200 ml	1	2	3	4	10	20	
Teaspoons	1	1	2	2	5	10	
Cotton wool, kg	1/2	1	11/2	2	5	10	
Adhesive tape, reels	1	1	1	2	3	6	
Hand soap, kg	2	4	6	8	20	40	
Boxes of soap for washing clothes	3	6	9	12	30	60	
1-litre bottle of cleaning solution (2% chlorine or 1–2% phenol)	1	1	1	1	2	4	

[a]Considering that less than 50% of the patients need IV rehydration.
[b]In case of multidrug resistance to above antibiotics, choose cefixime.

Developed by the WHO Global Task Force on Cholera Control.

APPENDIX 1: VIRAL HAEMORRHAGIC FEVER OUTBREAK CONTROL

Identify suspected cases of viral haemorrhagic fever (VHF).



Note: The above flowchart applies to the first steps for VHF outbreak investigation.

As soon as a VHF is suspected, VHF isolation precautions should begin. This will help to reduce the number of people exposed to the disease.

USE INFORMATION FROM PREVIOUS OUTBREAKS TO ASSESS RISK OF A VHF OUTBREK

Talk with the district or national surveillance officer about VHFs that have been reported in your area. Report suspected cases of VHF according to national surveillance guidelines to the corresponding health authorities.

Begin VHF isolation precautions

- Adapt VHF isolation precautions as needed.
- Designate the health officer who will coordinate VHF isolation precautions. As soon as a health care worker suspects a VHF, he or she should notify the health facility administrator and the VHF co-ordinator who will:
  - refer the patient to the isolation area and take the necessary steps to begin VHF isolation precautions as described below;
  - limit the number of health facility staff and visitors in the patient's room;
  - limit the use of invasive procedures and reduce the number of injectable medications.

Important: Between the time when VHF is suspected and the time when the patient is received in the isolation area, there is a risk for disease transmission from the patient's blood and other body fluids (stool, urine, vomitus). Prevent disease transmission to other patients, visitors and health staff in the waiting area by placing the suspected VHF patient apart from other patients. Make every effort to reduce this waiting time.

➢ *Reinforce standard universal precautions in the health centre/hospital*.

VHF isolation precautions

Isolation precautions can be started even if the diagnosis has not been laboratory-confirmed.

- Isolate the patient.
- Wear protective clothing in the isolation area, in the cleaning and laundry areas and in the laboratory. Wear a scrub-suit, gown, apron, two pairs of gloves, mask, headcover, eyewear and rubber boots.
- Clean and disinfect spills, waste and reusable equipment safely.*
- Clean and disinfect soiled linens and laundry safely.*
- Use safe disposal methods for non-reusable supplies and infectious waste.
- Provide information about the risk of VHF transmission to health facility staff. Reinforce the use of VHF isolation precautions with all health facility staff.
- Provide information to families and the community about prevention of VHFs and care of patients.

* Pour or soak in 0.5% chlorine solution; see *Guidelines for collection of specimens for laboratory testing* in this Toolkit (*Annex 7*).

See: Appendix 2: *Select the isolation area*, below.

Identify patient's contacts and travel history

Ask the patient (or a family member who can answer for the patient) questions on the following topics:
- Place where currently living.
- Other persons with the same symptoms in the family or village.
- Places the patient has visited in the past 3 weeks.

Use the answers to identify contacts. Provide contacts with information about VHF and when to seek care.

Specimen samples for laboratory confirmation

According to the suspected VHF, obtain specimens for confirmation of diagnosis. (See *Guidelines for collection of specimens for laboratory testing* in this Toolkit (*Annex 7*) for specific techniques for collecting blood and other specimens from suspected VHF cases and their method of transport).

All suspected cases should be reported and laboratory specimens given to the corresponding health authority (surveillance officer or WHO officer) or person responsible for coordinating epidemic control and transporting/shipping of specimens to the appropriate reference laboratory and for follow-up of results.

Alert health facility staff about specific risks for VHF transmission

- As soon as a VHF is suspected, alert the relevant health staff to begin using VHF isolation precautions. This applies especially to:
  - doctors or nurses providing direct patient care;
  - cleaning, laundry and waste disposal staff who clean and disinfect contaminated material and supplies;
  - laboratory staff who handle samples from the suspected VHF cases;
  - medical or support staff who prepare or handle the bodies of deceased VHF patients.

- Explain how VHF transmission can occur in the health facility and the risks to health facility staff. Remind the staff that VHF is a highly infectious disease. They must use VHF isolation precautions whenever they have contact with a VHF patient, the patient's blood or other body fluids, or contaminated supplies and equipment.

APPENDIX 2: SELECT THE TREATMENT ISOLATION AREA

Establish a barrier between the VHF patient and uninfected patients, other health facility staff and visitors.

Description

- A single room with an adjoining toilet or latrine.
- A separate building or ward that can be used for VHF patients only (especially if Ebola haemorrhagic fever is suspected, or if there is a large number of patients).
- An area in a larger ward that is separate and far away from other patients in the ward.

Important: There should be an isolated toilet, adequate ventilation and screened windows.

Place a security barrier around the isolation area and restrict access. Place signs around the isolation area clearly stating that access is restricted.

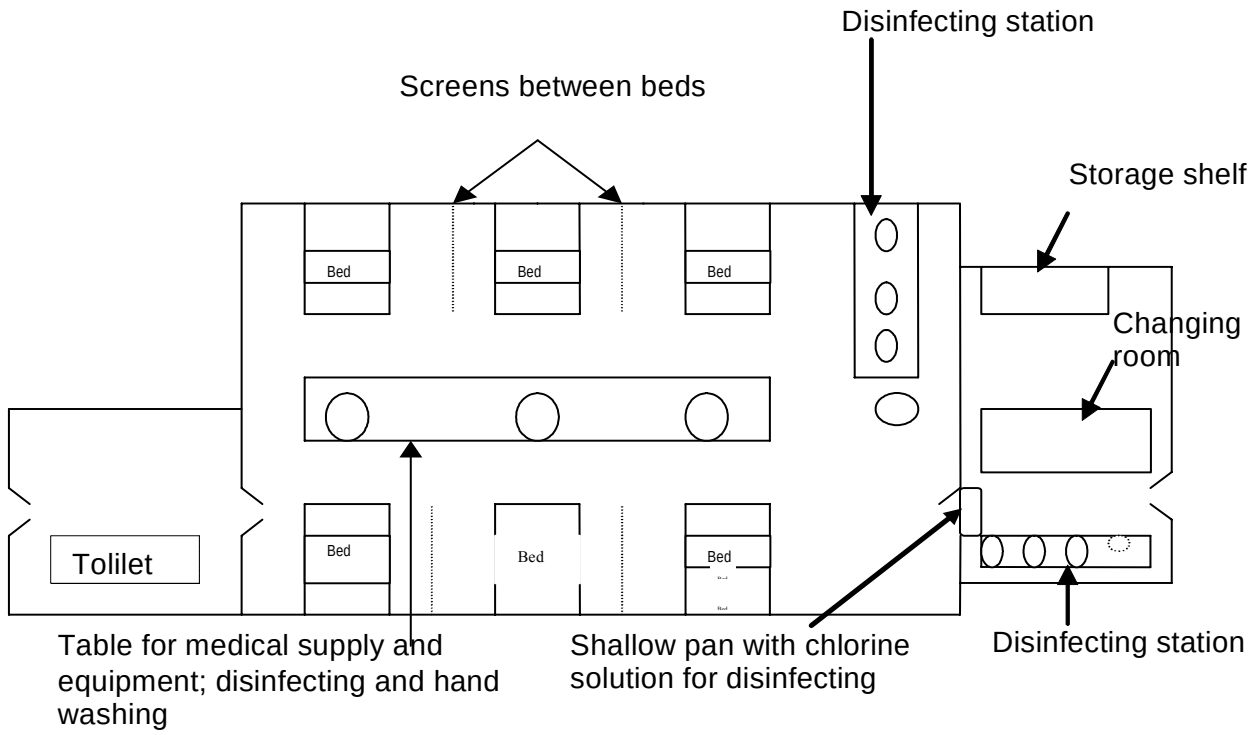
Set up changing rooms for staff providing patient care

One changing room is needed outside the patient isolation area. This is where health care workers will put on protective clothing. Contaminated clothing and supplies remain in the changing room until cleaning staff – trained to use VHF isolation precautions – take the VHF-contaminated items to the laundry or disposal site.

If there are family members who will assist with direct patient care, give them information and training about:

– the risk of VHF transmission and the reason for protective clothing;
– how to wear gloves, gowns and a mask;
– how to remove gloves, gowns and mask, and store or dispose of them safely.

FIGURE 2. EXAMPLE OF VIRAL HAEMORRHAGIC FEVER (VHF) TREATMENT ISOLATION AREA



APPENDIX 3: SAFE BURIAL PRACTICES

The bodies and body fluids of deceased VHF patients remain contagious for several days after death. Family and community members are also at risk if funeral practices involve touching and washing the body.

Prepare the body safely

The burial should take place as soon as possible after the body is prepared in the health facility. Health facility staff should:

– prepare the body safely;
– be aware of the family's cultural practices and religious beliefs, and help the family to understand why some practices cannot be observed because they place the family or others at risk for exposure and death.

To prepare the body in the health facility:

1. Wear protective clothing as recommended for staff in the patient isolation area. Use thick rubber gloves as the second pair (or outer layer) of gloves.
2. Spray the body and the area around it with a 0.5% chlorine solution.*
3. Place the body in a body bag (mortuary sack) and close it securely. Spray the body bag with a 0.5% chlorine solution.*
4. If a body bag is not available, wrap the body in two thicknesses of cotton cloth soaked with a 0.5% chlorine solution*. Then wrap the body in plastic sheeting. Seal the wrapping with plastic tape. Spray the body bag as in Step 3. Place the body in a coffin if one is available.
5. Transport the body to the burial site as soon as possible. Assign a health officer or a member of the health facility staff to accompany the body to ensure that the safety precautions continue to be observed during the journey.

Prepare burial site

- The grave should be at least 2 metres deep.
- Carefully explain to the family the reason for limiting attendance at the funeral ceremony to family only.

Disinfect the vehicle after transporting the body

- The staff member who disinfects the vehicle must wear protective clothing.
- Rinse the interior of the vehicle in which the body was carried with a 0.5% chlorine solution* and let it soak for 10 minutes.
- Rinse well with clean water and let the vehicle air-dry.

* See Appendix 8 of *Guidelines for collection of specimens for laboratory testing* in this Toolkit (*Annex 7).*

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COMMUNICABLE DISEASE TOOLKIT

SUDAN

6. CASE MANAGEMENT OF EPIDEMIC-PRONE DISEASES

World Health Organization

CONTENTS

1. BACILLARY DYSENTERY (SHIGELLOSIS)

Basic facts

- Bacillary dysentery is an acute bacterial disease involving the large and small intestines.
- It is the most important cause of acute bloody diarrhoea.
- Two-thirds of cases and most deaths occur in children aged under 10 years.
- Of the four *Shigella* serogroups (*S. dysenteriae, S. flexneri, S. sonnei and S. boydii*), *S. dysenteriae* type 1 (Sd1) causes the most severe disease and is the only cause of large-scale epidemics.

Shigella dysenteriae type 1:

- Most severe in young children, the elderly and malnourished individuals.
- Displaced populations are at high risk in situations of overcrowding and poor sanitation/water.
- Transmission is by the faecal–oral route from person-to-person and through contaminated food and water.
- Highly contagious: as few as 10–100 bacteria have caused disease in volunteers.
- Treatment is with antimicrobials, which reduce severity and duration of illness.
- Not usually associated with marked loss of fluid and electrolytes.
- Without prompt, effective treatment, the case-fatality rate may be as high as 10%.
- As infectious dose is low, shigellosis is associated with high secondary attack rates.

Clinical features

- Causes bloody diarrhoea, often associated with fever, abdominal cramps and rectal pain.
- Incubation period usually 1–3 days, but may be up to 1 week.
- Complications include sepsis, rectal prolapse, haemolytic uraemic syndrome, seizures.
- Is diagnosed by observing blood in a fresh stool specimen or asking the patient or mother of a child whether the stools are bloody.

Diagnosis

- Collect specimens from case with current bloody diarrhoea and onset of illness <4 days who has not been given antimicrobials for this illness.
- Fresh stools in sterile container must be kept at 4 °C and must reach the laboratory within 12 hours of collection. If fresh stools samples are not refrigerated they must reach the laboratory for culture.
- Where transport to the laboratory will take longer, Cary-Blair transport media must be used.
- Transport container should be well insulated with freezer packs or wet ice.
- Transport must not take more than 3 days.

Case management

Clinical case definition: acute bloody diarrhoea.

Laboratory criteria: Isolation of *Shigella dysenteriae* type 1 (Sd1) from stool samples.

Table 1. High-risk patients

• Children aged under 5 years, but especially infants, severely malnourished children and children who have had measles in the past 6 weeks
• Older children and adults who are obviously malnourished
• A patient who is severely dehydrated, has had a convulsion, or is seriously ill when first seen
• Adults aged 50 years or older

Standard treatment regimens:

A. Rehydrate with ORS or IV solution depending on severity, and monitor the hydration status frequently. (**See Appendix for assessment and treatment of diarrhoea and dehydration.**)

- Refer seriously ill or severely malnourished patients to hospital immediately.

B. Give antibiotics

- Antibiotics are essential and should be selected on the basis of susceptibility testing of the organisms grown from patients affected by the disease. The drugs must be effective against the local Sd1 strains.
- If an antimicrobial is effective, clinical improvement should be noted within 48 hours. If there is no improvement, treat with second-line drug, if available, for 5 days; otherwise, continue full 5-day course of first-line drug. Use only one of the following antibiotics:

Antibiotic	Dose	Children		Adults
	< 1 year	1–5 years	5–15 years	
Ciprofloxacin 500 mg	30 mg/kg divided 2 times/day for 3 days	½ tablet 2 times/day for 3 days	1 tablet 2 times/day for 3 days	1 tablet 2 times/day for 3 days

***Note**: Do not give antimicrobials that are known to be ineffective. When the supply of an effective antimicrobial is limited, priority should be given to high risk patients (see Table 1).*

Do not forget:

- In health facilities
  - strengthen sanitary and hygiene measures in general;
  - implement disinfection measures in wards.

- In affected areas
  - ensure access to safe water (adequate quality and quantity);
  - strengthen health education on hygiene and disinfection measures;
  - set up surveillance for early detection of cases and monitoring of outbreak.

See *Guidelines for outbreak control* in this Toolkit (Document 5) for organization of an emergency treatment centre (*Figure 1*), essential hygiene rules in a cholera treatment centre (*Table 4*), preparation and use of disinfectants (*Table 5*) and calculation of treatment supplies for dysentery (*Table 7*).

This section was developed by the WHO Global Task Force on Cholera Control.

2. CHOLERA

Basic facts

- Cholera is an acute bacterial enteric disease with profuse watery stool.
- It is caused by a Gram-negative bacillus, *Vibrio cholera*, which produces a powerful enterotoxin that causes copious secretary diarrhoea.
- Transmission is by the feacal–oral route. Infection results from ingestion of organisms in food and water, or from indirect person-to-person contamination (unwashed hands).
- Acute carriers, including those with asymptomatic or mild disease, are important in the maintenance and transmission of cholera.
- Cholera is asymptomatic in more than 90% of infected cases.
- Attack rates in displaced populations can be as high as 10–15%; in normal situations, estimated at 1–2%.
- Case–fatality rates are usually around 5% but have reached 40% in large outbreaks in refugee camps.
- With appropriate treatment (with ORS in most cases), the case-fatality rate can be reduced to 1%.

Clinical features

- Incubation period is 1–5 days.
- Onset of symptoms is abrupt, with copious watery diarrhoea, classic “rice-water” stool with or without vomiting.
- Fluid loss can lead to rapid and profound dehydration, low serum potassium and acidosis.
- Fever is unusual, except in children.
- Vomiting without associated nausea may develop, usually after the onset of diarrhoea.
- Severe dehydration leads to loss of skin turgor, malaise, tachypnoea and hypotension.

Early detection of cholera cases is important to ensure prompt treatment and reduction of environmental contamination. Cholera should be suspected when:

- a patient aged over 5 years develops severe dehydration from acute watery diarrhoea (usually with vomiting)
  or
- any patient aged over 2 years has acute watery diarrhoea in an area where there is an outbreak of cholera.

Diagnosis

- Fresh stools in sterile container if transport time is less than 2 hours.
- In alkaline peptone water if transport time is less than 24 hours.
- Cary-Blair transport media.
- Media previously cooled for 1 hour.
- Transport container well insulated.
- Transport within for 7–14 days after collection.

Case management

Clinical case definition: acute watery diarrhoea with or without vomiting, with or without severe dehydration, once cholera has been confirmed.

Laboratory criteria: Isolation of *Vibrio cholerae* O1 or O139 from stools

The prevention and treatment of dehydration are the mainstays of cholera management:

STEP 1 Assess for dehydration (see Appendix)
STEP 2 Rehydrate and monitor frequently
STEP 3 Maintain hydration: replace ongoing fluid losses until diarrhoea stops
STEP 4 Give oral antibiotics to patients with severe dehydration
STEP 5 Feed the patient:
– ensure normal intake of food as soon as possible;

– breastfeeding for infants and young children should continue.

Standard treatment regimens:

A. Rehydrate with ORS or IV solution depending on severity, and monitor the hydration status frequently. (See Appendix for assessment and treatment of diarrhoea and dehydration.)

- For severe dehydration, give IV fluid immediately to replace fluid deficit. Use Ringer's lactate or Hartmann's solution or, if not available, normal saline solution.
  Plain glucose solutions are ineffective and should not be used.

B. Give antibiotics for severe cholera cases only.

Antibiotic	**Dose**	**Children**			**Adults**	**Pregnant women**
		Under 1 year	1–5 years	5–15 years		
Erythromycin 250 mg	30 mg /kg divided, 4 times/day for 3 days	¼ tablet 4 times/day for 3 days	½ tablet 4 times/day for 3 days	1 tablet 4 times/day for 3 days	2 tablets 4 times/day for 3 days	2 tablets 4 times/day for 3 days
Doxycycline 100mg	300 mg single dose				3 tablets	

- **Antibiotic therapy is not essential** to the management of cholera. **Effective rehydration therapy is life-saving.** In emergencies, systematic administration of antimicrobials is justified only for severe cases and in situations where bed occupancy, patient turnover or stocks of IV fluids are expected to reach critical levels in respect of case management capacity.

- An antibiotic sensitivity profile of the outbreak strain must be available as soon as possible to decide on the possible choice of antibiotic. Only oral antimicrobials must be given, and only once the patient has been rehydrated (usually in 4–6 hours) and vomiting has stopped.

Do not forget:

- In health facilities
  - strengthen sanitary and hygiene measures in general
  - implement disinfection measures in cholera wards
  - implement special funeral practices:
    disinfect corpses with 2% chlorine solution;
    fill the mouth and anus of the corpse with cotton wool soaked with 2% chlorine solution;
    wash hands with soap after touching the corpse;
    disinfect the clothing and bedding of the deceased by stirring them in boiling water or by drying them thoroughly in the sun.

- In affected areas:
  - ensure access to safe water (adequate quality and quantity);
  - strengthen health education on hygiene, disinfection measures and food safety;
  - set up surveillance for early detection of cholera cases and monitoring of outbreak.

- Chemoprophylaxis and quarantine measures are not effective in containing the spread of cholera.

See *Guidelines for outbreak control* in this Toolkit (Annex 5) for organization of an emergency treatment centre (Figure 1), essential hygiene rules in a cholera treatment centre (Table 4), preparation and use of disinfectants (Table 5) and calculation of treatment supplies for cholera (Table 6).

This section was developed by the WHO Global Task Force on Cholera Control.

3. TYPHOID FEVER

Basic facts

- Typhoid fever is a serious systemic infection caused by the enteric bacillus *Salmonella enterica* serovar Typhi (*S.*Typhi)
- Transmission is via the faecal–oral route, mainly from ingestion of organisms in food and water contaminated by faeces and urine of patients and carriers, or indirectly from person-to-person (unwashed hands).
- 2–5% of infected cases remain carriers for several months, and are highly involved in the spread of the disease.
- Without proper treatment case-fatality rate is high (10–20%).
- With appropriate antibiotic therapy, case-fatality rate can be reduced to 1%.
- Relapses occur in 3–4% of cases.
- Some strains of *S.*Typhi are resistant to antibiotics.
- Mass immunization may be a valuable adjunct for the control of typhoid fever during a sustained, high-incidence epidemic.
- A parenteral vaccine containing the polysaccharide Vi antigen is the vaccine of choice for displaced populations; a single injection provides effective protection, and adverse reactions are minimal.

Clinical features

- Incubation period is usually 8–14 days, but may vary from 3 days to as much as 1 month.
- Mild or inapparent forms are common, especially in endemic areas, and present with low-grade fever and malaise.
- Severe symptoms begin with the sudden onset of sustained fever, severe headache, nausea and loss of appetite, sometimes accompanied by hoarse cough and constipation or diarrhoea.
- Complications of intestinal ulceration can include intestinal perforation or haemorrhage.

Diagnosis

- Isolation of *S.*Typhi from blood culture early after disease onset, or from stool culture after the first week.
- Because of limited specificity and sensitivity, serological tests are generally of little diagnostic value.

Case management

Clinical case definition: acute or insidious onset of sustained fever, headache, malaise, anorexia, relative bradycardia, constipation or diarrhoea and non-productive cough (but many mild and atypical infections also occur).

Laboratory criteria: isolation of relevant serovars of *S.*Typhi from stool or blood of patient.

Standard treatment regimens:

Rehydrate with ORS or IV solution, depending on severity. (See Appendix for assessment and treatment of diarrhoea and dehydration).

Give antibiotics

Antibiotics are essential and should be selected on the basis of susceptibility testing of the organisms grown from patients affected by the disease. Only one of the following antibiotics should be used:

Susceptibility	Antibiotic	Daily dose	Days
Fully sensitive	Chloramphenicol	50–75 mg/kg	14–21
	Amoxicillin	75–100 mg/kg	14
	Co-trimoxazole	8–40 mg/kg	14
Multidrug-resistant	Cefixime	15–20 mg/kg	7–14
	Azithromycin	8–10 mg/kg	7

Treatment of complications

Treatment for complications may include rest, diuretics, ionotropes, and anti-arrhythmic drugs for myocarditis, replacement blood components for bone marrow suppression and blood transfusion for the haemhorragic problems. Surgery is necessary in case of intestinal perforation.

Vaccination

Vaccination against typhoid fever during an outbreak should be considered: please contact the WHO Global Task Force on Cholera Control (e-mail: cholera@who.int).

Do not forget:

- In health facilities
  - strengthen sanitary and hygiene measures in general;
  - implement disinfection measures in wards;
  - implement special funeral practices.

- In affected areas
  - ensure access to safe water (adequate quality and quantity);
  - strengthen health education on hygiene and disinfection measures;
  - set up surveillance for early detection of cases and monitoring of outbreak.

See *Guidelines for outbreak control* in this Toolkit (Annex 5) for organization of an emergency treatment centre (Figure 1), essential hygiene rules in a cholera treatment centre (Table 4), preparation and use of disinfectants (Table 5) and calculation of treatment supplies for a typhoid outbreak (Table 8).

This section was developed by the WHO Global Task Force on Cholera Control.

4. EBOLA VIRAL HAEMORRHAGIC FEVER (VHF)

Basic facts:

- Ebola haemorrhagic viral fever (VHF) is an acute viral illness caused by the Ebola virus which belongs to the *Filovirus* group.
- It is transmitted from person to person by direct contact (spread) by droplets onto mucous membranes) or indirectly by infected blood, secretions, organs, semen and vomit. Under natural conditions, airborne transmission among humans has not been documented. Nosocomial infections have been frequent.
- The reservoir is not known, and is therefore difficult to evaluate the risk of transmission. The implementation of control measures can be difficult due also to cultural reasons, such as the custom of eating primate meat.

Clinical Features

- **Incubation period is usually for Ebola VHF is 2 to 21 days**
- Presentation may be very non specific. Initial symptoms include acute fever, diarrhoea that can be bloody (referred to as *diarrhée rouge* in francophone Africa) and vomiting. Headache, nausea and abdominal pain are common. Conjunctival injection, dysphagia and hemorrhagic symptoms (nosebleeds, bleeding gums, vomiting of blood, blood in stools, purpura) may further develop. Some patients may show a maculopapular rash on the trunk. Dehydration and significant wasting occur as the disease progresses.
- At a later stage, there is frequent involvement of the central nervous system, manifested by somnolence, delirium or coma.
- The case-fatality rate ranges from 50% to 90% according to the virus.

Case classification

Suspected: a case that is compatible with the clinical description.
Probable (in epidemic situation):

- Any person having had contact with a clinical case and presenting with acute fever, **or**
- Any person presenting with acute fever and 3 of the following: headache, vomiting/nausea, loss of appetite, diarrhoea, intense fatigue, abdominal pain, general or articular pain, difficulty in swallowing, difficulty in breathing, hiccoughs, **or**
- Any unexplained death

Confirmed: Any suspected or probable case that is laboratory-confirmed.

Contact (in epidemic situation): An asymptomatic person having had physical contact within the past 21 days with a confirmed or probable case or his/her body fluids (e.g. care for patient, participation in a burial ceremony, handling of potentially infected laboratory specimens.

Diagnosis

This can **only** be done in a laboratory of biosafety level 4 reference laboratory.

Specific diagnosis of VHF can be made in the following ways:

- isolating the virus from blood, urine or throat swabs and other tissues;
- Positive ELISA antigen detection or IgM capture, or
- Positive virus isolation (only in a laboratory of biosafety level 4), or
- Positive skin biopsy (immunohistochemistry), or
- Positive reverse transcriptase/polymerase chain reaction or immunohistochemistry (post-mortem diagnosis (PCR) with sequence confirmation.

The most common diagnostic test is the enzyme-linked immunosorbent assay (ELISA), which can detect IgM antibody (acute infection) and IgG antibody (recent infection) as well as the virus antigen.

Case management

There is no specific therapy currently available for filoviral infections

Supportive treatment includes the use of

- Analgesic drugs
- Antimicrobial drugs (to avoid secondary infections)
- Fluid replacement with careful maintenance of fluid and electrolyte balance, circulatory volume, blood pressure. Most of the fluid replacement should be done orally.
- oxygenation,
- treatment of any other complicating infection (e.g. malaria, measles)
- Mechanical ventilation, renal dialysis, and anti-seizure therapy may be required.

Remember: All medication should be given by the oral or intravenous route. Intramuscular and subcutaneous injections are contraindicated because of the risk of hematomas.

Implementation of barrier nursing practices is of crucial importance when managing VHF patients. In order to prevent secondary infections contact with the patient's lesions and body fluids should be minimized using standard isolation precautions:

- Isolation of patients
- Restriction of access to patients wards
- Use of protective clothing
- Safe disposal of waste
- Disinfection of all non-disposable supplies and equipment
- Safe burial practices

These can be implemented despite problems due to limited resources (see WHO/CDC. Infection control for viral hemorrhagic fevers in the African care setting. Geneva: WHO, 1998. WHO/EMC/EST/98.2)

Protective measures

Patients with probable or confirmed VHF should be isolated and cared for using **barrier-nursing techniques**. Isolation precautions to reduce the risk of transmission of Lassa fever in the health care setting should follow the guidelines developed by WHO/CDC. **Universal precautions** must be observed when handling specimens of blood or tissues, and when disposing of waste material, needles, and other sharp instruments.

See:
-"VHF outbreak control" in Guidelines for outbreak control, in this Toolkit (Annex 5)
-Infection Control for Viral Haemorrhagic Fevers in the African Health Care Setting, available online at: http://www.who.int/emc-documents/haem_fevers/whoemcesr982c.html
-"Prevention" in Section 7, HIV/AIDS in the Communicable Disease Profile of this Toolkit.
-ANNEX 8 in Guidelines for collection of specimens for laboratory testing, in this toolkit.

Hospital control

Basic barrier nursing methods (gloves, gowns and masks) are highly effective in preventing secondary spread of the infection. Strict isolation with rigorous barrier nursing should be combined with full medical care, to ensure the safety of the staff and survival of the patient.

Epidemics of the disease in health care institutions with poor hygiene standards can be dramatically amplified through contact with patients or body fluids from infected patients (blood, vomit, urine, stools, semen, saliva). The potential for explosive nosocomial infections constitutes the main threat to public health posed by the disease. Strict adherence to isolation precautions with all patients has been shown to reduce the risk of transmission, as was observed during the May to June 2004 Ebola hemorrhagic fever outbreaks in Yambio county, Southern Sudan. A total of 17 cases and 7 deaths of Ebola VHF were reported. Thirteen of the cases were laboratory-confirmed and 4 cases epidemiologically linked. The rapid containment of this outbreak was a tremendous success for the health authorities, WHO, and the international community involved in the control operations.

The following will help prevent explosive epidemics in areas potentially subject to Ebola disease:

- Social mobilization and health education of the community.
- Advance training of health workers on the use of isolation precautions, proper barrier nursing methods and the regular consistent practice of universal precautions.

5. MEASLES

Basic facts

- Measles is a highly communicable viral infection transmitted through airborne spread of respiratory droplets from person to person, or by direct contact with nasal and throat secretions of infected persons or via objects that have been in close contact with an infected person.
- It is a severe disease caused by the rubeola virus, which damages epithelial surfaces and the immune system.
- Measles can increase susceptibility to other infections such as pneumococcus and Gram-negative bacteria.
- It can lead to or exacerbate vitamin A deficiency, increasing the susceptibility to xerophthalmia, blindness and premature death.
- The most vulnerable age group is children aged between 9 months and 5 years in developing countries, but this depends on the immunization coverage rates.
- Deaths are mostly the result of complications such as pneumonia, croup and diarrhoea and are frequently associated with malnutrition.

Note: *While this section details diagnosis and case management of measles, immunization remains the most important strategy for measles control. Measles immunization campaigns are one of the highest priorities in displaced populations. The recommended age group for immunization is 6 months to 15 years, with vitamin A supplementation for children aged 6–59 months. Those vaccinated between the ages of 6 and 9 months must have another dose on reaching 9 months of age.*

Clinical features

- Incubation period from exposure to onset of fever is usually 10 days.
- Initial symptoms and signs are high fever, runny nose, coryza, cough, red eyes and Koplik spots (small white spots on the buccal mucosa).
- Characteristic erythematous (red) maculopapular (blotchy) rash appears on the third to seventh day, starting behind the ears and on the hairline and then spreading to the rest of the body.
- Temperature subsides after 3–4 days; the rash fades after 5–6 days.
- Measles is highly infectious from the start of the prodromal period until approximately 4–5 days after the rash appears.
- Case–fatality rates are estimated to be 3–5% in developing countries but may reach as much as 10–30% in displaced populations.

Complications

- Complications develop in 5–10% of measles cases.
- Complications occurring in the first week of illness, such as croup, diarrhoea and pneumonia, are usually due to effects of the measles virus and are rarely life-threatening.
- Later complications are usually due to secondary viral or bacterial infections – post-measles pneumonia, diarrhoea and croup are the most common life-threatening complications.
- Pneumonia: usually severe, Gram-negative or staphylococcal.
- Diarrhoea: either due to virus or to a secondary infection, e.g. *Shigella*.
- Malnutrition: precipitated by anorexia, stomatitis, fever, vomiting, diarrhoea and other complications.
- Stomatitis: compromises sucking and eating.
- Vitamin A deficiency: keratoconjunctivitis. Measles increases the need for vitamin A and often precipitates xerophthalmia.
- Encephalitis: caused by the measles virus itself, occurs on about the 5th day of the rash.
- Otitis media, croup.
- Blindness due to scarring, as a result of vitamin A deficiency and/or conjunctivitis.

Case management

Take a history from the mother and examine the child for the following:

Symptoms	Signs
Ability to take feeds of fluids	Nutritional status
Cough and difficult breathing	Breathing rate, chest indrawing, stridor
Diarrhoea or blood in stools	Dehydration and fever
Sore mouth, eyes or ears	Mouth ulcers, sore and discharging ears and eyes, white spots on eyes Level of consciousness

Case management of uncomplicated measles – health centre

Most children will have uncomplicated measles and require supportive care as outpatients. Good supportive care can improve a child's outcome. Isolation of patients with measles is not indicated in emergency situations. All children with measles in these settings should have their nutritional status monitored and be enrolled in a feeding programme if necessary.

Nurse the child in a shaded and well ventilated area, which is generally more comfortable – sunlight can hurt the eyes, and a cool environment can help keep the body temperature down.

- Control the fever by tepid sponging and giving paracetamol.
- Maintain good hydration: treat diarrhoea with ORS.
- Observe closely for complications.
- Give prophylaxis against xerophthalmia: vitamin A on day 1 and day 2

	Day 1	Day 2
Infants <6 months	50 000 IU	50 000 IU
Infants 6–11 months	100 000 IU	100 000 IU
Children >11 months	200 000 IU	200 000 IU

- Maintain adequate protein–calorie intake: tell mothers to give frequent small meals.
- Continue breastfeeding.
- Provide supplementary feeding, if available. The diet must be soft, with a high calorie density so that small portions go a long way. Unless in the form of egg, protein is unlikely to be eaten – *remember the child has a sore mouth and poor appetite.*
- Do not admit children with measles to *general* feeding centres until after infectious period.
- If there are high numbers of cases, it may be necessary to set up a small unit for children with measles, as these children and their mothers need considerable supportive care.
- Use antimicrobials only when indicated.
- Active case-finding during epidemic if practical (home visits).

Case management of complicated measles – hospital

- Control fever, provide nutritional support and vitamin A therapy as for uncomplicated measles.
- Antimicrobials should be given only if there is a specific indication such as pneumonia, otitis media or dysentery.
- Prophylactic antimicrobials should be given to children at significant risk of secondary bacterial infection – such as children with severe malnutrition, HIV infection or xerophthalmia. A broad-spectrum antibiotic such as ampicillin or co-trimoxazole should be used.
- Pneumonia: cough and rapid breathing (40 breaths/minute or more if aged over 1 year; 50 breaths/minute if aged under 1 year). Give an antibiotic such as ampicillin, amoxicillin or co-trimoxazole. If the child's condition does not improve after 24–48 hours, change the antibiotic to an antistaphylococcal drug such as cloxacillin or chloramphenicol.
- Diarrhoea: three or more loose or watery stools in 24 hours. Assess for associated dehydration. If there is blood in the stool, the child has dysentery. The commonest cause of dysentery is *Shigella* (see "Bacillary dysentery/Shigellosis" for case management).

- Eye problems: the major eye problems in measles are conjunctivitis or keratitis, and corneal damage due to vitamin A deficiency. Red and watery eyes are the result of conjunctivitis (inflammation of the conjunctiva): no treatment is necessary.
- Sticky eyes or pus in the eyes are caused by a secondary bacterial infection: clean the eyes at least three times a day with cooled boiled water, using cotton wool or a clean cloth. Use tetracycline ointment three times a day for 7 days. NEVER use steroid eye ointments. Ensure that vitamin A has been given; if there is vitamin A eye disease, a third dose must be given 4 weeks later.

6. MENINGITIS

Basic facts

- Meningitis is an acute inflammation of the meninges that can be caused by bacteria or viruses.
- Transmission is through direct contact with respiratory droplets.
- Large outbreaks of meningitis are mainly due to meningococcus (*Neisseria meningitidis*, serogroups A, C and W*)*.
- *N. meningitidis* also causes meningococcal septicaemia – this is a less common but very severe disease with acute fever, purpura and shock.
- *N. meningitidis*, *Streptococcus pneumoniae* and *Haemophilus influenzae* account for 80% of all cases of bacterial meningitis.
- Viral meningitis is rarely serious and may be due to a number of viruses such as Coxsackievirus or Enterovirus.
- Displaced populations are at increased risk of meningitis due to overcrowding, poor hygiene and poor access to health care.
- Epidemics in refugee camps have mainly been due to *N. meningitidis* serogroup A.
- 80% of cases of meningococcal meningitis occur in those aged under 30 years.
- Without appropriate treatment, case–fatality rates in meningococcal meningitis can be as high as 50%. This can be reduced to 5–15% by correct treatment.
- Vaccines are available against *N. meningitidis* serogroups A, C and W135 and are very effective in controlling epidemics. In rapid mass campaigns, vaccination can contain an outbreak within 2–3 weeks. For individuals aged over 2 years, vaccine efficacy is 90% one week after injection.

Diagnosis

- Ask about: sudden onset of intense headache, fever, nausea, vomiting, photophobia, stiff neck.
- Examine for:
  - meningeal rigidity, i.e. neck stiffness
  - lethargy, delirium, coma
  - purpura – characteristic sign of meningococcal septicaemia
  - symptoms of shock – low blood pressure.
- In a child <1 year, classic signs are rare, look for:
  - fever, diarrhoea, vomiting, drowsiness
  - convulsions
  - bulging fontanelle

Lumbar puncture is necessary to determine whether acute meningitis is bacterial and should be done as soon as meningitis is suspected, before starting antimicrobials. In bacterial meningitis, CSF is usually cloudy or purulent (but may be clear or bloody). Basic laboratory examination consists of white cell count (WCC), protein and Gram stain.

Bacterial meningitis if:
WCC measurement: >1000 cells/mm^3 (<3 in normal CSF) with >60% polymorphs
Protein: >0.80 g/litre (<0.60 g/litre in normal CSF)
Gram stain: Gram-negative diplococci in 80% of cases not previously treated

Differential diagnosis of bacterial meningitis
Viral meningitis: do lumbar puncture and examine CSF

Case management

- Bacterial, particularly meningococcal, meningitis is potentially fatal and is a medical emergency.
- Viral meningitis is rarely serious and requires supportive care, but a lumbar puncture is necessary to differentiate from bacterial meningitis.
- Admit all suspected meningitis cases to hospital for diagnosis and case management.
- Perform lumbar puncture and give antimicrobials immediately without waiting for results.
- Do not delay treatment with antimicrobials if lumbar puncture cannot be done.

Table 1. Initial empirical antimicrobial therapy for presumed bacterial meningitis

Age group	Probable pathogen	Antimicrobial – first choice	Alternative
In epidemic situations: all age groups	*N. meningitidis*	Oily chloramphenicol	Ampicillin Ceftriaxone or Cefotaxime Co-trimaxozole Benzylpenicillin
In non-epidemic situations: adults children >5 years	*N. meningitidis* *S. pneumoniae*	Benzylpenicillin or oily chloramphenicol	Ampicillin Ceftriaxone or Cefotaxime Co-trimaxozole
children 1 month – 5 years	*H. influenzae* *S. pneumoniae* *N. meningitidis*	Ampicillin or amoxicillin Chloramphenicol	Ceftriaxone or Cefotaxime
neonates	Gram-negative bacteria Group B streptococci *Listeria*	Ampicillin and gentamicin	Ceftriaxone or Cefotaxime Chloramphenicol

- IV benzylpenicillin, ampicillin, ceftriaxone or cefotaxime is recommended for bacterial meningitis; however, ceftriaxone and cefotaxime are very expensive.
- In patients who cannot be given drugs IV, oral administration is acceptable but higher doses are necessary.
- During large epidemics in refugee/displaced populations, a single IM dose of oily chloramphenicol has been used.
- In meningococcal septicaemia with purpura and shock, treat shock by restoring blood volume; give IV dexamethasone to reduce cerebral oedema.
- Chemoprophylaxis of contacts is not recommended in emergency situations.
- Supportive therapy: maintain hydration and adequate nutrition.
- Treat convulsions by giving diazepam IV or rectally.
- Nurse patients in a shaded and well-ventilated area. The unconscious or semiconscious patient should be nursed on his or her side. Turning every 2–3 hours can prevent pressure sores.

Table 2. Antimicrobials to treat bacterial meningitis

Agent	Route	Daily dose for adults	Daily dose for children	Duration (days)	Cost[a]
Benzylpenicillin	IV	3–4 Million Units 4–6 times	400 000 U/kg	>4	low
Ampicillin/ amoxicillin	IV	2–3 g twice	250 mg/kg	>4	medium
Amoxicillin	Oral	2–3 g twice	250 mg/kg	>4	high
Chloramphenicol	IV	1 g 2–3 times	100 mg/kg	>4	medium
Chloramphenicol (oily)	IM	3 g single dose	100 mg/kg	1–2	low
Cefotaxime	IV	2 g twice	250 mg/kg	>4	very high
Ceftriaxone	IV	1–2 g once or twice	50–80 mg/kg	>4	low
Ceftriaxone	IM	1–2 g, single dose	50–80 mg/kg	1–2	low
Co-trimoxazole	IV/IM	2 g SMZ[b] twice	100 mg/kg	>4	medium
Co-trimoxazole	Oral	2 g SMZ[b] twice	100 mg/kg	>4	low
Sulfadiazine	IV	1 g six times	200 mg/kg	>4	low

[a] Guide to cost of full treatment: low <US$ 10; medium US$ 10–50; high US$ 50–250, very high >$250.
[b] Sulfamethoxazole.

7. YELLOW FEVER

Basic facts

- Yellow fever is a viral haemorrhagic fever transmitted by mosquitoes infected with the yellow fever virus. The incubation period is 3–7 days.
- Mosquitoes are infected by feeding on patients in the first 3–4 days of illness, when the virus is circulating in the blood.
- The disease is untreatable, and case–fatality rates in severe cases can exceed 50%.
- Yellow fever can be prevented by immunization with the 17D yellow fever vaccine. The vaccine is safe, inexpensive and reliable – a single dose provides protection against the disease for at least 10 years and possibly for life.
- Any person who is not immunized against yellow fever is at risk for the disease.
- An outbreak of yellow fever is defined as at least one confirmed case.
- In an outbreak situation, the target population for emergency immunization is the general population living or working in the same area as the patient. If initial resources are limited, the primary target population is children aged 9 months up to 14 years.

Clinical features

- An *acute phase* lasting 4–5 days and presenting with:
  - sudden onset of fever
  - headache or backache
  - muscle pain
  - nausea
  - vomiting
  - red eyes (injected conjunctiva).

  Because jaundice may not be present in less severe (or mild) cases of yellow fever, this phase of the disease can be confused with other diseases that also present with fever, headache, nausea and vomiting. The less severe cases are often non-fatal.

- A temporary *period of remission* follows the acute phase in 5–20% of cases and lasts for up to 24 hours.

- A *toxic phase* can follow the period of remission and present with:
  - jaundice
  - dark urine
  - reduced urine production
  - bleeding from the gums or nose or in the stool
  - vomiting blood
  - hiccups
  - diarrhoea
  - slow pulse in relation to fever.

WHO case definition for yellow fever surveillance

Suspected case: an illness characterized by an acute onset of fever followed by jaundice within 2 weeks of onset of the first symptoms AND one of the following: bleeding from the nose, gums, skin or gastrointestinal tract OR death within 3 weeks of the onset of illness.
Confirmed case: a suspected case that is confirmed by laboratory results or linked to another confirmed case or outbreak.
Outbreak: an outbreak of yellow fever is at least one confirmed case.

Diagnosis

- Laboratory analysis of blood or tissue samples (usually liver) is needed to confirm a case of yellow fever. Two blood samples must be taken.
- Yellow fever is confirmed if laboratory results show:
  - isolation of the yellow fever virus, or
  - presence of yellow fever-specific IgM, or
  - a fourfold or greater rise in serum IgG levels between the acute and convalescent serum samples,

  OR
  - positive postmortem lever histopathology, or
  - detection of yellow fever antigen in tissues by immunohistochemistry, or
  - detection of yellow fever virus RNA genomic sequences in blood or tissues.

Note: Liver samples are taken from fatal cases only.

Case management

- No specific treatment is available for yellow fever. In the toxic phase, supportive treatment includes therapies for treating dehydration and fever. In severe cases, death can occur 7–10 days after onset of the first symptoms.
- For fever: give paracetamol.
- For dehydration: give ORS solution or IV fluids depending on the assessment of dehydration.
- For restlessness: give diazepam.
- For malaria: give an antimalarial recommended for your area.
- For bacterial infections: give antibiotics recommended for your area.

APPENDIX

ASSESSMENT AND TREATMENT OF DIARRHOEA

Table A1. Assessment of diarrhoeal patients for dehydration

First assess your patient for dehydration			
	PLAN A	**PLAN B**	**PLAN C**
1. Look at general condition	Well, alert	***Restless, irritable***	***Lethargic or unconscious; floppy***
eyes[a]	Normal	Sunken	Very sunken and dry
tears	Present	Absent	Absent
mouth and tongue[b]	Moist	Dry	Very dry
thirst	Drinks normally, not thirsty	***Thirsty, drinks eagerly***	***Drinks poorly or not able to drink***
2. Feel skin pinch[c]	Goes back quickly	***Goes back slowly***	***Goes back very slowly***
3. Decide	The patient has *no signs of dehydration*	If the patient has two or more signs, including at least one ***sign*** there is *some dehydration*	If the patient has two or more signs, including at least one ***sign*** there is *severe dehydration*
4. Treat	Use Treatment Plan A	Weigh the patient if possible and use Treatment Plan B	Weigh the patient and use Treatment Plan C **URGENTLY**

[a] In some infants and children the eyes normally appear somewhat sunken. It is helpful to ask the mother whether the child's eyes are normal or more sunken than usual.
[b] Dryness of the mouth and tongue can also be palpated with a clean finger. The mouth may always be dry in a child who habitually breathes through the mouth. The mouth may be wet in a dehydrated patient owing to recent vomiting or drinking.
[c] The skin pinch is less useful in infants or children with marasmus (wasting) or kwashiorkor (severe malnutrition with oedema) or in obese children.

Source: *The treatment of diarrhoea: a manual for physicians and other senior health workers.* Geneva, World Health Organization, 1995 (WHO/CDR/95.3).

Treatment plan A: to treat diarrhoea at home

Use this plan to teach the mother to:

- continue to treat her child's current episode of diarrhoea at home; and
- give early treatment for future episodes of diarrhoea.

Explain the three rules for treating diarrhoea at home.

A. Give the child more fluids than usual to prevent dehydration

- Use recommended home fluids. These include ORS solution, food-based fluids (such as soup, rice water and yogurt drinks) and plain water. Use ORS solution as described in the box below.

Note: If the child is under 6 months of age and not yet taking solid food, give ORS solution or water rather than food-based fluid.

- Give as much of these fluids as the child will take. Use the amounts shown below for ORS as a guide.
- Continue giving these fluids until the diarrhoea stops.

B. Give the child plenty of food to prevent malnutrition

- Continue to breastfeed frequently.
- If the child is not breastfed, give the usual milk.
- If the child is 6 months or older, or already taking solid food:
  - also give cereal or another starchy food mixed, if possible, with pulses, vegetables and meat or fish; add one or two teaspoonfuls of vegetable oil to each serving;
  - give fresh fruit juice or mashed banana to provide potassium;
  - give freshly prepared foods; cook and mash or grind food well;
  - encourage the child to eat: offer food at least six times a day; and
  - give the same food after diarrhoea stops, and give an extra meal each day for 2 weeks.

C. Take the child to the health worker if he or she does not get better in 3 days or develops any of the following:

- many watery stools
- repeated vomiting
- marked thirst
- eating or drinking poorly
- fever
- blood in the stool

Children should be given ORS solutions at home if:

- they have been on Treatment Plan B or C;
- they cannot return to the health worker if the diarrhoea gets worse; or
- if it is national policy to give ORS to all children who see a health worker for diarrhoea.

If the child is to be given ORS solution at home, show the mother how much ORS to give after each loose stool and give her enough packets for 2 days.

Age	Amount of ORS solution to be given after each loose stool	Amount of ORS solution to provide for use at home
Under 24 months	50–100 ml (¼ – ½ cup)	500 ml/day
2–10 years	100–200 ml (½ – 1 cup)	1000 ml/day
10 years or older	as much as wanted	2000 ml/day

- Describe and show the amount to be given after each stool, using a local measure.

Show the mother how to mix and to give ORS

- Give a teaspoonful every 1–2 minutes for a child aged under 2 years.
- Give frequent sips from a cup for older children.
- If the child vomits, wait 10 minutes. Then give the solution more slowly (for example, a spoonful every 2–3 minutes).
- If diarrhoea continues after the ORS packets are used up, tell the mother to give other fluids as described in rule A above or return for more ORS.

Treatment plan B: to treat dehydration

Table A2. Approximate amount of ORS solution to give in the first 4 hours

	Age[a]					
	<4 months	**4–11 months**	**12–23 months**	**2–4 years**	**5–14 years**	**15 years +**
Weight	0 – <5 kg	5–7.9 kg	8–10.9 kg	11–15.9 kg	16–29.9 kg	30 kg +
In ml	200–400	400–600	600–800	800–1200	1200–2200	2200–4000

[a] Use the patient's age only when you do not know the weight. The approximate amount of ORS required (in ml) can also be calculated by multiplying the patient's weight (in grams) by 0.075.

- If the child wants more ORS than shown, give more.
- Encourage the mother to continue breastfeeding.
- For infants aged under 6 months who are not breastfed, also give 100–200 ml clean water during this period.

Observe the child carefully and help the mother give ORS solution.

- Show her how much solution to give the child.
- Show her how to give it – a teaspoonful every 1–2 minutes for a child aged under 2 years, frequent sips from a cup for an older child.
- Check from time to time to see whether there are problems.
- If the child vomits, wait 10 minutes and then continue giving ORS, but more slowly; for example, a spoonful every 2–3 minutes.
- If the child's eyelids become puffy, stop the ORS and give plain water or breast milk. Give ORS according to Plan A when the puffiness is gone.

After 4 hours, reassess the child using the assessment chart, then select Plan A, B or C to continue treatment.

- If there are no signs of dehydration, shift to Plan A. When dehydration has been corrected, the child usually passes urine and may also be tired and fall asleep.
- If signs indicating some dehydration are still present, repeat Plan B but start to offer food, milk and fruit juice as described in Plan A.
- If signs indicating severe dehydration have appeared, shift to Plan C.

If the mother must leave before completing Treatment Plan B:

- show her how much ORS to give to finish the 4-hour treatment at home;
- give her enough ORS packets to complete rehydration, and for 2 more days as shown in Plan A;
- show her how to prepare ORS solution; and
- explain to her the three rules in Plan A for treating her child at home:
  - to give ORS or other fluids until diarrhoea stops
  - to feed the child
  - to bring the child back to the health worker, if necessary.

Treatment plan C: to treat severe dehydration quickly

Follow the arrows. If the answer is "yes" go across. If "no" go down.

Can you give intravenous (IV) fluids immediately?

Yes →

Start IV fluids immediately. If the patient can drink, give ORS by mouth while the drip is set up. Give 100 ml Ringer's lactate solution per kg of body weight (or, if not available, give normal saline), *divided as follows*:

Age	First give 30 ml/kg in:	Then give 70 ml/kg in:
Infants (under 12 months)	1 hour*	5 hours
Older	30 minutes*	2½ hours

* Repeat once if radial pulse is still very weak or undetectable.

- Reassess the patient every 1–2 hours. If hydration is not improving, give the IV drip more rapidly.
- Also give ORS (about 5 ml/kg per hour) as soon as the patient can drink: usually after 2–4 hours (infants) or 1–2 hours (older patients).
- After 6 hours (infants) or 3 hours (older patients), evaluate the patient using the assessment chart. Then choose the appropriate Plan (A, B or C) to continue treatment.

No ↓

Is IV treatment available nearby (within 30 minutes)?

Yes →

- Send the patient immediately for IV treatment.
- If the patient can drink, provide the mother with ORS solution and show her how to give it during the trip.

No ↓

Are you trained to use a nasogastric tube for rehydration?

Yes →

- Start rehydration by tube with ORS solution: give 20 ml/kg per hour for 6 hours (total of 120 ml/kg).
- Reassess the patient every 1–2 hours:
  - if there is repeated vomiting or increased abdominal distension, give the fluid more slowly;
  - if hydration is not improved after 3 hours, send the patient for IV therapy.
- After 6 hours, reassess the patient and choose the appropriate treatment plan.

No ↓

Can the patient drink?

Yes →

- Start rehydration by mouth with ORS solution, giving 20 ml/kg/hour for 6 hours (total of 120 ml/kg).
- Reassess the patient every 1–2 hours:
  - if there is repeated vomiting, give the fluid more slowly;
  - if hydration is not improved after 3 hours, send the patient for IV therapy.
- After 6 hours, reassess the patient and choose the appropriate treatment plan.

No ↓

Urgent: send the patient for IV or nasogastric treatment.

If possible, observe the patient for at least 6 hours after rehydration to be sure the mother can maintain hydration giving ORS solution by mouth. If the patient is older than 2 years and there is cholera in the area, give an appropriate oral antibiotic after the patient has become alert.

WHO/CDS/2005.26

COMMUNICABLE DISEASE TOOLKIT

SUDAN

7. GUIDELINES FOR COLLECTION OF SPECIMENS FOR LABORATORY TESTING

World Health Organization

INTRODUCTION

There is a high risk of communicable disease outbreaks in emergency situations. Outbreaks must be recognized and controlled rapidly in order to minimize their impact. **Effective containment of an outbreak depends on**:

- **early detection and reporting of suspect cases**
- **rapid epidemiological investigation**
- **rapid laboratory confirmation of the diagnosis**
- **implementation of effective control measures.**

Rapid identification of the causative agent and the likely source or mode of transmission is essential. The initial investigation involves two important processes: collection of information on suspect cases and collection of clinical specimens for laboratory diagnosis. **Successful laboratory confirmation of a disease depends on**:

- **advance planning**
- **collection of appropriate and adequate specimens**
- **correct packaging of specimens and rapid transport to an appropriate laboratory**
- **the ability of the laboratory to carry out the diagnostic tests**
- **proper biosafety and decontamination procedures to reduce the risk of further spread of the disease.**

The purpose of this document is to ensure that the correct specimens are collected, packaged and transported in a safe and standardized manner during a field investigation of an outbreak in Sudan or its neighbouring countries.

This document is adapted for emergency situations from *Guidelines for the collection of clinical specimens during field investigation of outbreaks*. Geneva, World Health Organization, 2000 (WHO/CDS/CSR/EDC/2000.4).

1. Planning for specimen collection

Once a suspected outbreak has been detected and reported, an epidemiological investigation must be quickly organized. The materials and procedures required for efficient specimen collection and their transport to the laboratory for testing are outlined below.

1.1 Define the possible causes of the outbreak

An assessment of current clinical and epidemiological information is the starting point for considering the potential etiology of the outbreak. The historical knowledge of regional endemic and epidemic diseases, as well as their seasonality, further defines the possible causes. Since a variety of infectious agents can present with a similar clinical picture, the outbreak should be approached in a syndromic manner to obtain the differential diagnosis. One or more specimen types may be required to define the cause of the outbreak.

1.2 Decide which clinical specimens are required to confirm the cause of the outbreak

After defining the clinical syndrome and suspect pathogen(s), decide on the clinical specimens to be collected for appropriate laboratory diagnosis.

1.3 Laboratory for specimen testing

In the event of an outbreak, WHO will coordinate the transport of specimens and follow up on result of laboratory tests.

1.4 Collecting the specimens

For stools, the health worker should collect the sample, place in cold-box and inform WHO. Transport to the laboratory should be done as soon as possible. For CSF, the admitting physician should conduct the lumbar puncture and obtain the sample. Blood samples should be taken by the health worker.

2. Specimen collection and processing

Investigation should start as early as possible after a suspected outbreak has been notified. Specimens obtained in the acute phase of the disease, preferably before administration of antimicrobial drugs, are more likely to yield detectable concentrations of antibody, antigen or infective pathogen. Before beginning specimen collection, explain the procedure to the patient and relatives. When collecting the specimen, avoid contamination and take a sufficient quantity of material (as guided by the laboratory tests). Follow the appropriate precautions for safety during collection and processing of specimens.

2.1 Labelling and identification of specimens

In an outbreak investigation, the information contained in the case investigation and laboratory request forms is collected along with the specimen. Each patient should be assigned a unique identification number by the collection team. This is the link between the laboratory results on the line listing form, the specimens and the patient, which guides further investigation and response to the outbreak. This unique identification number and the patient's name should be present on all specimens, epidemiological data forms and the laboratory request and should be used as a common reference.

2.2 Labelling specimen container/slide

Labels must always be used. The label should be very clearly written and permanently affixed to the specimen container.
It should contain the:

- patient name
- unique identification number
- specimen type and date and place of collection
- name or initials of specimen collector.

2.3 Case investigation and laboratory forms

A case investigation form should be completed for each patient at the time of specimen collection. The original case investigation form remains with the investigation team, and should be kept together for analysis and later reference. A laboratory form must also be completed for each specimen. The epidemiological and clinical data gathered in the investigation can later be easily tied to the laboratory results for analysis.

The form includes:

- patient information: name, age (or date of birth), sex, complete address.
- clinical information: date of onset of symptoms, clinical and immunization history, risk factors, antimicrobials taken before specimen collection
- laboratory information: acute or convalescent specimen, other specimens from the same patient.

The form records the date and time when the specimen is received and the name of the person collecting the specimen.

3. Storage of specimens

To preserve bacterial or viral viability in specimens for microbiological culture or inoculation, they should be placed in appropriate media and stored at recommended temperatures. These conditions must be preserved throughout transport to the laboratory and will vary according to the nature of specimens and pathogens (sensitivity to desiccation, temperature, nutrient and pH) and the time required to transport the specimens to the laboratory.

Many specimens taken for viral isolation are viable for 2 days if maintained in type-specific media at 4–8 °C. Freeze these specimens in accordance with expert advice, as infectivity may be altered.

Specimens for bacterial culture should be kept in appropriate transport media at the recommended temperature. This ensures bacterial viability while minimizing overgrowth of other microorganisms. With the exception of CSF, urine and sputum, most specimens may be kept at ambient temperature if they will be processed within 24 hours. For longer delays, storage at 4–8 °C is advisable except in the case of particularly cold-sensitive organisms, such as *Shigella, Meningococcu*s and *Pneumococcus*. Longer delays are not advisable as the yield of bacteria may fall significantly.

Specimens for antigen or antibody detection may be stored at 4–8 °C for 24–48 hours or at –20 °C for longer periods. Sera for antibody detection may be stored at 4–8 °C for up to 10 days. Although not ideal, sera stored at room temperature may still be useful for antibody testing even after prolonged periods (weeks). Therefore, sera that have been collected should not be discarded simply because no refrigeration facilities are available.

APPENDIX 1: LABORATORIES FOR CONFIRMATION OF PRIORITY DISEASES IN SUDAN

Suspected organism/disease	Laboratory
Vibrio cholera O1: stool	• WHO accredited laboratories in the region. • Institut Pasteur, Paris, France for confirmation
Shigella dysenteriae type 1: stool	• WHO accredited laboratories in the region. • Institut Pasteur, Paris, France for confirmation
Meningitis: cerebrospinal fluid	• Rapid tests for meningococcal serotypes A, C, W135 available with some NGOs (e.g. MSF). • WHO accredited laboratories in the region. • Transport for culture to Institut Pasteur, Paris, France.
Measles, yellow fever (2 tubes): blood, serum	• WHO accredited laboratories in the region.
Acute flaccid paralysis: stool	• WHO accredited laboratories in the region.
Haemorrhagic fevers: blood, urine	• WHO accredited laboratories in the region.

APPENDIX 2: BLOOD SPECIMEN COLLECTION

Blood and separated serum are the most common specimens taken in outbreaks of communicable disease. Venous blood can be used for isolation and identification of the pathogen in culture and by inoculation, or separated into serum for the detection of genetic material (e.g. by polymerase chain reaction), specific antibodies (by serology), antigens or toxins (e.g. by immunofluorescence). For the processing of most specimens for diagnosis of viral pathogens, serum is preferable to unseparated blood except where otherwise directed. When specific antibodies are being assayed, it is often helpful to collect paired sera, i.e. an acute sample at the onset of illness and a convalescent sample 1–4 weeks later. Blood can also be collected by finger-prick for the preparation of slides for microscopy or for absorption onto special filter-paper discs for analysis. Whenever possible, blood specimens for culture should be taken before antibiotics are administered to the patient.

Venous blood samples

Materials for collection

- Skin disinfection: 70% alcohol (isopropanol, ethanol) or 10% povidone iodine, swabs, gauze pads, adhesive dressings.
- Disposable latex or vinyl gloves.
- Tourniquet, vacutainer or similar vacuum blood collection devices, or disposable syringes and needles.
- Vacutainer or sterile screw-cap tubes (or cryotubes if indicated), blood culture bottles (50 ml for adults, 25 ml for children) with appropriate media.
- Labels and indelible marker pen.

Method of collection

Full infection control measures must be taken, with gowns, gloves, masks and boots for suspected viral haemorrhagic fever such as Lassa fever or Ebola (see *Appendix 7*).

- Place a tourniquet above the venepuncture site. Disinfect the tops of blood culture bottles.
- Palpate and locate the vein. The venepuncture site **must** be meticulously disinfected with 10% povidone iodine or 70% alcohol by swabbing the skin concentrically from the centre of the venepuncture site outwards. Let the disinfectant evaporate. Do not repalpate the vein. Perform venepuncture.
- If using conventional disposable syringes, withdraw 5–10 ml of whole blood from adults, 2–5 ml from children and 0.5–2 ml from infants. Using aseptic technique, transfer the specimen to relevant capped transport tubes and culture bottles. Secure caps tightly.
- If using a vacuum system, withdraw the desired amount of blood directly into each transport tube and culture bottle.
- Remove the tourniquet. Apply pressure to site until bleeding stops, and apply dressing.
- Label the tube, including the unique patient identification number, using indelible marker pen.
- Do not recap used sharps. Discard directly into the sharps disposal container.
- Complete the case investigation and the laboratory request forms using the same identification number.

Handling and transport

- Blood specimen bottles and tubes should be transported upright and secured in a screw-cap container or in a rack in a transport box. They should have enough absorbent paper around them to soak up all the liquid in case of spillage.
- For serum samples (e.g. measles, yellow fever, HIV), the blood cells must be separated from serum. Let the clot retract for 30 minutes, then centrifuge at 2000 rpm for 10–20 minutes and pour off serum. If no centrifuge is available, place sample in refrigerator overnight (4–6 hours) and pour off the serum for transport in a clean glass tube.
- Do **not** attempt this in case of suspected viral haemorrhagic fever unless you are a clinician/laboratory technician experienced in management of the disease. Full protection and infection control measures must be taken (see *Appendix 7*).
- If the specimen will reach the laboratory within 24 hours, most pathogens can be recovered from blood cultures transported at ambient temperature. Keep at 4–8 °C for longer transit periods, unless the bacterial pathogen is cold-sensitive.

APPENDIX 3: CEREBROSPINAL FLUID (CSF) SPECIMEN COLLECTION

The specimen must be taken by a physician or a person experienced in the lumbar puncture procedure. CSF is used in the diagnosis of viral, bacterial, parasitic and fungal meningitis/encephalitis.

Materials for collection

Lumbar puncture tray that includes:

- Sterile materials: gloves, cotton wool, towels or drapes.
- Local anaesthetic, needle, syringe.
- Skin disinfectant: 10% povidone iodine or 70% alcohol.
- Two lumbar puncture needles, small bore with stylet.
- Six small sterile screw-cap tubes and tube rack.
- Water manometer.
- Microscope slides and slide boxes.
- Trans-Isolate media if available (must be kept at 4–8 °C *while in storage*; allow to reach room temperature before introducing the CSF).

Method of collection

- As only experienced personnel should be involved in the collection of CSF samples, the method is not described in this document. CSF is collected directly into the screw-cap tubes. If the samples will not be transported immediately, separate tubes should be collected for bacterial and viral processing.
- If Trans-Isolate media is available, first ensure that the media has reached to room temperature, draw the collected CSF from the sterile tube and inject into the vacuum-sealed Trans-Isolate bottle. The bottle must be kept for at least 3 days at more than 25 °C to allow incubation.

Handling and transport

- In general, specimens should be delivered to the laboratory and processed as soon as possible.
- CSF specimens for bacteriology are transported at ambient temperature, generally without transport media. They must never be refrigerated as these pathogens do not survive well at low temperatures. If Trans-Isolate medium is available, follow the instructions on the packaging precisely.
- CSF specimens for virology do not need transport medium. They may be transported at 4–8 °C for up to 48 hours or at –70 °C for longer periods.

APPENDIX 4: FAECAL SPECIMEN COLLECTION

Stool specimens are most useful for microbiological diagnosis if collected soon after onset of diarrhoea (for viruses <48 hours and for bacteria <4 days) and preferably before the initiation of antibiotic therapy. If required, two or three specimens may be collected on separate days. Stool is the preferred specimen for culture of bacterial, viral and parasitic diarrhoeal pathogens. Rectal swabs showing faeces may also be taken from infants. Rectal swabs are not useful for the diagnosis of viruses.

Materials for collection

- Tubes with Cary-Blair transport medium.
- Clean, dry, leak-proof screw-cap container and tape if Cary-Blair transport medium is not available.
- Appropriate bacterial transport media for transport of rectal swabs from infants (ideally Cary-Blair).
- Parasitology transport pack: 10% formalin in water, polyvinyl isopropyl alcohol (PVA).

Method of collecting a stool specimen

If Cary-Blair transport medium is available:

- Place sterile swab in freshly passed stool to allow it to soak up stool.
- Place swab in the Cary-Blair transport medium inside the tube.
- Break off the top part of the stick without touching the tube and tighten the screw-cap firmly.
- Label the specimen tube.

If Cary-Blair transport medium is not available, collect freshly passed stool, 5 ml liquid or 5 g solid (pea-size), in a container. Label the container.

Method of collecting a rectal swab from infants

- Moisten a swab in sterile saline.
- Insert the swab tip just past the anal sphincter and rotate gently.
- Withdraw the swab and examine to ensure that the cotton tip is stained with faeces.
- Place the swab in sterile tube/container containing the appropriate transport medium.
- Break off the top part of the stick without touching the tube, and tighten the screw-cap firmly.
- Label the specimen tube.

Handling and transport

- For suspected **shigella** cases, stool specimens should be transported in a cold-box at 4–8 °C. Bacterial yields may fall significantly if specimens are not processed within 1–2 days of collection. *Shigella* is particularly sensitive to elevated temperatures. If transport medium is not available, do not allow the specimen to dry – add a few drops of 0.85% sodium chloride solution.
- For suspected **cholera** cases, take 10-20 stool samples to confirm the epidemic.
    - Stool samples should be taken with a rectal swab and transported in sterile Cary Blair medium. The sample needs to reach the laboratory within 7 days; refrigeration is **not** necessary. Cary Blair medium can be stored at ambient temperature for 1–2 years (should not be dried out or discoloured).
    - If Cary Blair transport medium is not available, a cotton-tipped rectal swab can be soaked in liquid stool, placed in a sterile plastic bag, tightly sealed, and sent to the laboratory within 2 *hours* OR strips of blotting or filter paper can be soaked with liquid stool and placed in a sealed tube or bag with 2–3 drops of normal saline (NaCl 9%). Refrigeration is **not** necessary.
- Specimens to be examined for parasites should be mixed with 10% formalin or PVA, 3 parts stool to 1 part preservative. Transport at ambient temperature in containers sealed in plastic bags.

APPENDIX 5: RESPIRATORY TRACT SPECIMEN COLLECTION

Specimens are collected from the upper or lower respiratory tract, depending on the site of infection. Upper respiratory tract pathogens (viral and bacterial) are found in throat and nasopharyngeal specimens. Lower respiratory tract pathogens are found in sputum specimens. Culture of certain organisms, such as *Legionella*, is difficult, and diagnosis is best based on the detection of antigen excreted in the urine.

When acute epiglottitis is suspected, no attempt should be made to take throat or pharyngeal specimens since these procedures may precipitate respiratory obstruction. Epiglottitis is generally confirmed by lateral neck X-ray, but the etiological agent may be isolated on blood culture.

Materials for collection

- Transport media – bacterial (TransAmies) and viral (Cellmatics)
- Dacron and cotton swabs
- Tongue depressor
- Flexible wire, calcium alginate-tipped swab (for suspected pertussis)
- Nasal speculum (for suspected pertussis – not essential)
- Suction apparatus or 20–50-ml syringe
- Sterile screw-cap tubes, and wide-mouthed clean sterile jars (minimum volume 25 ml)

Upper respiratory tract specimens

Method of collecting a throat swab

- Hold the tongue down with the depressor. Use a strong light source to locate areas of inflammation and exudate in the posterior pharynx and the tonsillar region of the throat behind the uvula.
- Rub the area back and forth with a Dacron or calcium alginate swab. Withdraw the swab without touching cheeks, teeth or gums and insert into a screw cap tube containing transport medium.
- Break off the top part of the stick without touching the tube, and tighten the screw cap firmly.
- Label the specimen containers.
- Complete the laboratory request form.

Method of collecting nasopharyngeal swabs (for suspected pertussis)

- Seat the patient comfortably, tilt the head back and insert the nasal speculum.
- Insert a flexible calcium alginate/Dacron swab through the speculum parallel to the floor of nose without pointing upwards. Alternatively, bend the wire and insert it into the throat and move the swab upwards into the nasopharyngeal space.
- Rotate the swab on the nasopharyngeal membrane a few times, remove it carefully and insert it into a screw-cap tube containing transport medium.
- Break off the top part of the stick without touching the tube, and tighten the screw-cap firmly.
- Label the specimen tube, indicating left or right side.
- Complete the laboratory request form.
- Repeat on the other side.

Lower respiratory tract specimens

Method of collecting sputum

- Instruct the patient to take a deep breath and cough up sputum directly into a wide-mouthed sterile container. Avoid saliva or postnasal discharge. Minimum volume should be about 1 ml.
- Label the specimen containers.
- Complete the laboratory request form.

Handling and transport

- All respiratory specimens except sputum are transported in appropriate bacterial/viral media.
- Transport as quickly as possible to the laboratory to reduce overgrowth by commensal oral flora.

- For transit periods up to 24 hours, transport bacterial specimens at ambient temperature and viruses at 4–8 °C in appropriate media.

APPENDIX 6: URINE SPECIMEN COLLECTION

Material for collection

- Sterile plastic cup with lid (50 ml or more).
- Clean, screw-top specimen transport containers ("universal" containers are often used).
- Gauze pads.
- Soap and clean water (or normal saline) if possible.
- Labels and indelible marker pen.

Method of collection

- Give the patient clear instructions to pass urine for a few seconds, and then to hold the cup in the urine stream for a few seconds to catch a mid-stream urine sample. This should reduce the risk of contamination from organisms living in the urethra.
- To reduce the risk of contamination from skin organisms, the patient should be directed to avoid touching the inside or rim of the plastic cup with the skin of the hands, legs or external genitalia. Tighten the cap firmly when finished.
- For hospitalized or debilitated patients, it may be necessary to wash the external genitalia with soapy water to reduce the risk of contamination. If soap and clean water are not available, the area may be rinsed with normal saline. Dry the area thoroughly with gauze pads before collecting the urine.
- Urine collection bags may be necessary for infants. If used, transfer urine from the urine bag to specimen containers as soon as possible to prevent contamination with skin bacteria. Use a disposable transfer pipette to transfer the urine.
- Label the specimen containers.

Handling and transport

- Transport to the laboratory within 2–3 hours of collection. If this is not possible, do not freeze but keep the specimen refrigerated at 4–8 °C. Keeping the specimen refrigerated will reduce the risk of overgrowth of contaminating organisms.
- Ensure that transport containers are leak-proof and tightly sealed.

APPENDIX 7: SAMPLE COLLECTION FOR VIRAL HAEMORRHAGIC FEVER (VHF) INVESTIGATION

All invasive procedures and investigations should be minimized until the diagnosis of VHF is confirmed or excluded. Only the specific diagnostic samples needed should be obtained from acutely ill humans.

Other routine blood samples should be avoided when investigating a case of VHF.

The blood samples should be kept in their original tube (sealed, sterile, dry tubes, monovette or vacutainer type).

Do not attempt to separate serum or plasma from blood clots in the field – this is highly risky in case of VHFs. If these procedures are needed they should be performed at the reference laboratory.

Each collected sample must be marked with "high risk". Labels prepared in advance for both specimens collected and laboratory request forms should bear the name of the patient, the date of collection and a coded link to the corresponding record of the case.

Precautions for sampling

In addition to basic safety precautions, certain other specific precautions and additional safety equipment are essential when investigating cases of VHF to protect skin and mucous membranes against the pathogens.

Blood specimens should be taken by a doctor or nurse experienced in the procedure. Urine samples should also be handled carefully: a 20-ml syringe may be used to transfer urine from a bedpan to the specified container.

Protective clothing should always be worn when handling specimens from suspected VHF cases:

- protective gown
- waterproof protective apron
- two pairs of latex gloves
- particulate filter face mask
- goggles
- rubber boots.

Method of collection

- Observe all the basic safety precautions when obtaining specimens samples from suspected VHF cases.
- For taking blood samples, it is advisable to use a vacuum blood sampling system (Monovette or vacutainer); however, you may use the equipment and procedure you are most familiar with to avoid the risk of accidents or spills.
- Withdraw 5–10 ml of whole blood from adults, 2–5 ml from children and 0.5–2 ml from infants, directly into the transport tube (blood sample tube).
- Avoid the use of disposable alcohol swabs to apply pressure to venepuncture wounds; it is advisable to use dry cotton wool balls or gauze swabs.
- After taking the sample, the blood sample tube should be externally disinfected by wiping with 0.5% hypochlorite solution (see *Appendix 8*).

Removing protective clothing

- When the procedure is finished, remove the apron. Before removing the outer pair of gloves, wash your hands with soap and water and rinse them in 0.5% hypochlorite solution (see *Appendix 8*) for 1 minute.
- Keep the inner gloves on while removing goggles, mask, anything used to cover the head and the external gown; before removing boots soak, them in 0.5% hypochlorite solution). Finally, remove the gloves and the inner gown. Then wash your hands thoroughly with soap and water and disinfect them with 70% isopropyl alcohol or povidone iodine.

- Dispose of all protective clothing, gloves and materials in a plastic bag and incinerate everything.
- Remember never to recap used sharps. Discard directly into a sharps disposal container for later incineration.

Handling and transport of samples from suspected VHF cases

Particular care to prevent external contamination of specimen containers during specimen collection is critical.

A triple packaging system is used:

- The blood sample tube should be transported upright and secured in a leak-proof secondary container with a screw-on cap and sufficient absorbent material to absorb all the contents should leakage occur. Ensure that the cap is screwed tight and the container labelled (specimen record). The secondary container should be externally disinfected by wiping with 0.5% hypochlorite solution (see *Appendix 8*).

- Specimen data forms, letters and information that identifies or describes the specimen and also identifies the shipper and receiver should be taped to the outside of the secondary container.

- The secondary container is then placed in a third container – the transport box. The outer part of the transport box should be clearly marked with the biohazard symbol and should bear an address label that clearly identifies the specimen, the shipper and the receiver (see section 2.2 above).

If the blood sample cannot be processed the same day, ice packs must be placed in the transport box in order to keep the sample cold (4–8 °C). Whole blood samples should not be frozen.

***Note**: All materials needed for the sample handling and transport are included in the "Specimen transport module" of the Outbreak investigation kit in this Toolkit (Annex 8).*

APPENDIX 8: CHEMICAL DISINFECTANTS

Chlorine is the recommended disinfectant for use in field outbreak investigations. An all-purpose disinfectant should have an available chlorine concentration of 0.1% (= 1 g/litre = 1000 ppm); a stronger solution of 0.5% (= 5 g/litre = 5000 ppm) should be used in situations such as suspected Lassa fever and Ebola virus outbreaks.

In preparing appropriate dilutions, it is important to remember that different products have different concentrations of available chlorine. The manufacturer may provide appropriate instructions for the preparation of solutions with the above concentrations. Otherwise, the guidelines provided below may be used. Chlorine solutions gradually lose strength, and so fresh solutions must be prepared daily. Clear water should be used because organic matter destroys chlorine.

Commonly used chlorine-based disinfectants include:

Sodium hypochlorite

Commercial liquid bleaches, such as household bleach (e.g. Chlorox, *eau de javel*), generally contain 5% (50 g/litre or 50 000 ppm) available chlorine.

To prepare a 0.1% chlorine solution: make a 1 in 50 dilution, i.e. 1 part bleach in 49 parts water, to give a final concentration of available chlorine of 0.1%. (For example, add 20 ml of bleach to approximately 1 litre of water.)

To make a 0.5% chlorine solution: make a 1 in 10 dilution, i.e. 1 part bleach in 9 parts water, to give a final concentration of available chlorine of 0.5%. (For example, add 100 ml of bleach to 900 ml water.)

Chloramine powder

While the bleach solution described above may satisfy all disinfection needs, chloramine powder may prove convenient for disinfecting spills of blood and other potentially infectious body fluids. It may also be useful under field conditions because of ease of transport. It contains approximately 25% available chlorine.

In addition to its use for spills, chloramine powder may be used to prepare liquid chlorine solutions. The recommended formula is 20 g of chloramine powder in 1 litre of clean water.

Decontamination of surfaces

Wear an apron, heavy-duty gloves and other barrier protection if needed, and wipe surfaces clean with an absorbent material. Disinfect surfaces by wiping with a 1:10 dilution of household bleach, and then incinerate all absorbent material in heavy-duty garbage bags.

Decontamination of blood or body fluid spills

Spills should be very liberally sprinkled with chloramine granules to absorb the liquid, and left for at least 30 minutes. If chloramine powder is not available, absorbent materials may be used to soak up most of the fluid before disinfection with 0.5% liquid bleach. These absorbent materials must then be disinfected in bleach before disposal.

Sterilization and reuse of instruments and materials

In a field outbreak situation, it is not advisable to consider sterilization and reuse of any instruments or materials. Sterilization techniques are therefore not required, and are not described in this document.

Disinfection of hands

The principal means of disinfecting hands is thorough washing with soap and water. If available, commercial hand disinfectants such as chlorhexidine or povidone iodine may be used.

WHO/CDS/2005.26

COMMUNICABLE DISEASE TOOLKIT

SUDAN

8. OUTBREAK INVESTIGATION KIT

World Health Organization

OUTBREAK INVESTIGATION KIT

Item	Unit	Quantity/kit
1. Basic consumables module		
Cotton wool, 100%, surgical quality	roll of 500 g	
Ballpoint pen		5
Pencil		5
Eraser		5
Felt-tip pen (waterproof)		5
Marking pen, water-resistant ink, black and blue		5
Notebook (A4, hard cover, squared paper)		5
Labels (blank, self-adhesive)	series	5
Ruler		5
Calculator		5
Scissors		5
Thermometer		5
Torch light		5
Sealing tape	roll	5
Normal saline (0.9%)	500 ml	5
Sharps container for disposal of needles and syringes, of about 3 litres capacity.		5
Chlorine granules, 500 mg / container		5
2. Common consumables for collection of all specimens		
Gauze swabs, 10 x 10 cm, 100% cotton, 12-ply, 17-thread, sterile	100 pieces/box	5
Disinfecting swabs, impregnated with 70% isopropyl alcohol	100 pieces/box	5
Microscope slides, 76 x 26 mm, cut edges	50 pieces/box	5
Cover glasses, 22 x 22 mm	1000/box	5
Storing box for slides, wooden frame, for 25 slides each	10 boxes/pack	5
Universal containers, 70 ml, 55 x 44 mm, reliable sealing and polyethylene cap, machine sterile with standard label	1000/pack	5
Braunoderm (alcohol + PVP-IOD) for surgical scrub, against bacteria, fungi, viruses (incl. hepatitis B and HIV)	1 litre/cont'r	5
Povidone iodine solution	500-ml/cont'r	5
Disinfecting solution for hands		5
3. Blood module		
Blood lancets, sterile, disposable	pack of 200	5
Monovettes (orange cap, 10 ml)	pack of 100	1
Monovettes (red cap, EDTA, 3 ml)	pack of 100	1
Needles for Monovettes 21G	pack of 100	1
Needles for Monovettes 23G	pack of 100	1
Butterfly needles for blood culture 21G	pack of 100	1
Disposable soft transfer pipettes	pack of 1000	1
Racks for blood tubes		5
Adhesive bandages (small)	pack	5
Blood culture bottles (Hemoline performance DUO, children)	12 vials/pack	5
Blood culture bottles (Hemoline performance diphasic)	12 vials/pack	5
Tourniquets with clip		5

Item	Unit	Quantity/kit
4. Respiratory module		
Tongue depressor	pack of 100	5
Flexible wire calcium alginate-tipped swab (for pertussis)	pack of 100	1
Syringe for suction, 50–60-ml, with catheter tip	pack of 60	2
Transport swabs with TransAmies transport medium	pack of 1000	1
Virus transport medium (Cellmatics)	pack of 50	1
5. Urine module		
Urine container with boric acid, with screw cap, 30 ml (sterile)	400/pack	1
6. Stool module		
Rectal swabs for adults		25
Rectal swabs for infants		25
Stool collection tubes with spoon	pack of 400	1
Tubes with Cary-Blair transport medium		100
7. CSF module		
Sterile cotton swab	100/pack	5
Bottle with Trans-isolate media		100
Spinal needle, 25G x 3.5	25/box	5
Spinal needles, 23G x 3.5	25/box	5
Needle for transfer into medium, 21G	100/box	
Microtube 2.0 ml, with mouth screw cap and skirted base	50/bag	
Local anaesthetics (lidocaine 2%, 2 ml), 25G needle, 5-ml syringe		100
8. Self-protection module		
Disposable surgical gowns		10
Disposable surgical face masks	50 pieces/box	5
Disposable gloves: sizes S, M, L	100 pieces/box	5
Goggles		10
Face mask		10
Disposable surgical caps, size M	50 pieces/box	5
Rubber surgical boots	Pair, size 42	5
Disposable impermeable shoe cover, length 38 cm	100 pieces/bag	5
Impermeable aprons, 90 cm x 112 cm		5
Visors/face-shields		5
9. Specimen transport module		
Specimen carrier (cool-box)		5
Icepacks	set of 24	5
Microcentrifuge tube rack		5
Complete combination packaging for infectious substances, BioPack 2 with 1.5-litre BioJar		5
CL-4 thermal control unit, polystyrene box set in fibreboard case with all labels and instructions		5